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Original Articles.

ON THE MECHANISM OF BRAIN INJURIES.*

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Preliminary Considerations. Various Theories of Concussion.

The question as to whether or not it is possible to have fatal injury to the head, in which there is no appreciable gross lesion to the brain substance directly, or through its membranes, so called concussion, has long been discussed by writers on this subject.

By the older authors, the celebrated case, recorded so graphically by Littré (1†) now nearly two centuries ago, was accepted as decisive of the possibility of such a thing, but being almost solitary, it was by some looked upon as the exception which proved the rule. Although the case most quoted, however, Littré's was not the only one of alleged fatal concussion, Sabatier (2), Boyn (3) and Mourier (4), have all left records of similar cases. In a more sceptical age these classic records have been somewhat severely criticised, and the light of a more advanced physiology and pathology, with the information derived from more carefully

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† These figures refer to the corresponding numbers in the Bibliography at the end of the paper.

conducted *post-mortem* examinations, has thrown serious doubt on the accuracy of the observations on which the conclusions were based.

Sir Prescott Hewett (5) leads the attack against the theory of Littré, based on his oft quoted case, and concludes that "it has still to be demonstrated that death can result from mere concussion." Holmes (7), Bryant (8), McEwen (9), and Savory (10) in this country, and Neudorfer (11), Nancrede (12) and Fano (13) abroad, are, in the main, with Hewett; and so far as my experience, based upon *post-mortem* examinations, as well as a large number of experimental observations goes, I must agree with them.

The school which supports the old view is neither so strong, in point of numbers, nor so powerfully armed with statistics as is that of its opponents; but it includes men whose opinion is of sufficient weight to give them the standing of an influential minority. Huguenin (14), Hutchinson (15), as well as some others, believe that death may result from concussion without gross lesion. Mr. Hutchinson admits that lesions exist, but holds that they are to be looked upon "as an index of the violence of the shake, and not as causes of death, nor perhaps even as serious complications;" and Mr. Savory, while denying the occurrence of death without lesions, admits that "the effects must be due to the shock of the violence rather than to the lesions."

This raises the question, What is the ultimate cause of the symptoms in so-called cases of concussion of the brain? Is it, for example, as the old writers supposed, that the brain is thrown into a state of vibration which causes for the time being a suspension of its functions, as was held by Bruns (17), Liston (18) and Miller (19), on more or less purely theoretical grounds; or are the multiple capillary hæmorrhages on which Fano (13), Nélaton (21) and Rokitansky (22) laid such stress, the important causal pathological factors; or again, must we look to some profound vascular disturbance in the nervous centres as the "*fons et origo mali?*"

The Vibration Theory as stated by the late Professor Miller of Edinburgh, is as follows:—"Under the impulse of a blow or fall the brain must sustain more or less vibration if the cranium

remain entire. It is a pulpy organ, which exactly fills a nearly spherical bony cavity, whose parietes are elastic in a considerable, though very variable, degree; and if these parietes sustain any sudden change of shape, their contents must sustain a corresponding amount of compression, as any alteration in the shape of a spherical cavity must lessen its capacity. Whenever any external force impinges on the cranium with sufficient violence, it must be flattened at the point of impact, and expanded in some other direction; but these changes are, in virtue of the very cause whence they originate, of but momentary duration; the point primarily flattened by the compressing force immediately resumes its original shape, which is necessarily followed by a corresponding return of the expanded portion of the cranium to its previous dimensions. These oscillations may occur several times in rapid succession, their number and extent depending on the elasticity of the cranium, and direction of the force applied. In concussion then, the entire brain sustains a series of vibrations and momentary compressions varying in number and amount in every imaginable degree in different cases."

These views are supported by Bruns, who says that: "the vibrations permeate the brain as a whole, as well as its individual component parts." Gama (23) and Alquié believed that while the brain elements remained unaffected, the brain itself might be knocked from side to side of the cranium; but the results of these observers must be received with due caution, as their investigations were carried out on an artificially constructed apparatus, which could only imperfectly imitate the exceedingly delicate anatomical arrangements of the human brain.

There are several points in the vibration theory, thus enunciated, to which exception must be taken.

In the first place, the brain in its normal state, whether or not it be a "pulpy organ," does not exactly fill the cavity. There is everywhere a slight, and in some places a considerable space between it and the bone, filled by the cerebro-spinal fluid, a fact of great pathological as well as anatomical importance.

Nor is the cranium, I submit, by any means a "spherical cavity." It is so far removed from a sphere, that the laws of physics which apply to cavities of that shape do not affect it. It has been shown over and over again that the capacity

of the cranium is in constant proportion to its size, any increase in the quantity of one element, *e.g.*, blood, being compensated for by an escape of another, *e.g.*, cerebro-spinal fluid, and *vice versâ*. And it is by an escape of one or other, or both of these fluids that a depression of the parietes is able to diminish the contents of the skull, which, by the mere fact of being depressed, has had its size diminished, and therefore proportionately its capacity.

That this depression is not permitted by any appreciable diminution in size of the brain itself, is shown by the fact that it requires the pressure of a whole atmosphere to reduce a body of the consistence of the brain by $\frac{1}{25000}$th of its volume, and in very few head injuries is the pressure anything like so great, Duret (24). This theory also leaves out of account the fact that the hemispheres are hollowed out by the ventricles, and that before pressure could directly diminish the volume of the brain substance these cavities would have to be obliterated.

Other anatomical arrangements in the shape of the falx cerebri and tentorium cerebelli, etc., seem to me so to fix the brain in position that any considerable displacement of it would be evidenced by lacerations, due to its impinging on these tight resistent bands.

Taking all these points into consideration, I do not think there is sufficient evidence to show that the vibration theory is tenable.

The next theory, namely, that the symptoms of concussion are to be accounted for by the multitudes of capillary hæmorrhages so often found in the cerebral substance, has as its chief supporters, MM. Fano and Nélaton, and with them in the main Stromeyer (25) agrees, although he differs from them as to when these extravasations take place, the former assigning them to the primary stage, the latter to the stage of reaction. Chassaignac and Sanson are also disposed to look upon these lesions as sufficient to account even for death in fatal cases.

Drs. Bright (20) and Blandin (16) do not go so far. They believe that "in addition to minute extravasations, patches of contusion of the cerebral substance are necessary to account for death, if not for less serious symptoms."

On the other hand Huguenin (14) and McEwen (9) do not believe that these scattered lesions are sufficient to account for the phenomena, and with them M. Duret (24) is at one. He says that he "sought as carefully as possible at the autopsies and found only small lesions quite insufficient to explain so grave phenomena."

Mr. Jonathan Hutchinson argues that these lesions cannot be the cause of the symptoms and of death, because many cases with worse lesions, not only live but have no symptoms.

Unfortunately for this theory multiple hæmorrhages are by no means constant, many cases occurring in which none are to be found. Besides, the fact that the symptoms of concussion, in many cases, so soon pass off, argues against their being due to lesions, which from their very nature, could only disappear after a period, at least much longer than is required for the cessation of all evidences of concussion.

Neither of the theories just considered, namely (1) that of physical mechanical vibration; and (2) multiple petechial hæmorrhages seems to be sufficient to account for the varied and general symptoms of concussion.

Is that of derangement of the cerebral circulation any more acceptable?

When we consider the general character of the symptoms, how widely spread they are, affecting every function and organ in the body, from the prefrontal lobes with their highly specialised actions, to the least important and simplest mechanical muscle, we comprehend how almost impossible it would be for any single focal lesion, however diffuse, to be held responsible for all of these. And if we bear in mind the close resemblance which this group of symptoms bears to other conditions—such as cardiac syncope, reflex syncope from pain, or severe mental shock, where the cause is acknowledged to be a disturbance of the circulation of the brain—we are induced to accept this explanation of the phenomena, as at least a more workable theory than either of the other two. Most modern writers accept this view of the subject.

Some, such as Savory (10), McEwen (9), Le Gros Clark

(6), and others, define concussion as a form of "shock," which is only modified in so far as the hurt has expended its force on the brain.

Mr. Hutchinson (15) regards "anæmia of the brain" as being "the proximate cause of the phenomena of concussion," but whether this condition is produced by contraction of the arteries, or by diminution of the venous tension leading to congestion, and so an "oxygenless" condition of the cortex, he does not give an opinion.

Huguenin (14) accounts for the anæmia by the blood being squeezed out of the vessels by momentary compression of the brain substance, whose normal function depends on a proper supply of oxygenated blood, and which so suffers from thus being temporarily deprived of it, that its functions are suspended for a period varying in proportion to the severity of the injury as indicated by the amount of vascular disturbance.

It is doubtful if simple mechanical compression of the brain substance is the cause of the anæmia, as we have already seen that a much greater force is necessary to accomplish this than is almost ever applied in simple cases of concussion, and Nothnagel (28) has tried to explain the contraction of the blood-vessels on the assumption that it is the reflex effect of strong irritation of peripheral nerves. He does not explain, however, how this state of contraction can be kept up reflexly for several days, as is sometimes the case in concussion. Jolly (26), Rigel (27), and Frey (29) have failed to reproduce these symptoms by reflex irritation of vaso-motor nerves, although Goltz has produced a reflex paralysis of the vessels by injury to the head, which supports Fischer in his belief that this is the true explanation of the symptoms.

I propose now to consider what clinical and experimental proof we have of the existence of such a vascular disturbance.

Influence of Concussion on the Heart and General Circulation.

The experiments and observations on animals to which I am about to refer are described in detail in another part of

my paper. Unless the injury was intended to kill the animal instantaneously, full chloroform anæsthesia was produced before the experiment was commenced.

In the first case, that of a rabbit before the injury was inflicted, when the animal was in an excited state, the pulse was 200, and only countable with difficulty. After a severe occipital blow had been delivered however, there was marked slowing of the pulse. During the first minute following the blow no cardiac impulse could be felt whatever. During the second minute the action was very irregular—for the first fifteen seconds the pulsations were at the rate of forty to the minute, but this was followed by a rapid succession of small beats which were quite uncountable, but which, if counted, would of course have raised the number of beats per minute considerably. This irregularity, however, was evidently due to the action of respiration, at all events, it was synchronous with long-drawn, deep inspirations, and if we assume the causal relationship between the two, then the heart *per se* was certainly slowed, and that in a marked degree, and whether we make this assumption or not we can safely say that the circulation was seriously interfered with.

In another experiment on a rabbit, before a moderately severe frontal blow had been administered the heart was beating regularly and at the rate of 190 per minute. During the first minute after the blow the heart was acting so tumultuously as to be quite uncountable, and continued to do so for the next two minutes. At the end of four minutes, however, it was again regular, vigorous, and acting at the rate of 130. The animal was deeply under chloroform.

In a third rabbit, from which the cerebro-spinal fluid was rapidly aspirated under chloroform, the heart was from the first slow and feeble.

In a fourth experiment where the pulse before the blow was given was 180, rapid and small, two minutes after, it was down to 120, and described as full and bounding.

M. Duret (24) has demonstrated in an experiment carried out with the object of ascertaining accurately the heart rate following a blow to the head, that there is a

period during which the pulse is very slow, full and bounding, and this period he finds is synchronous with a disturbance and slowing of the respiration.

The experiment was performed on a dog with the following results :—

Before the blow the pulse was	...	...	...	100	p.	min.
1st minute after blow	...	...	...	14	,,	
2nd ,, ,,	...	...	...	16	,,	
3rd ,, ,,	...	...	...	12	,,	
4th ,, ,,	...	...	...	12	,,	

This stage is followed by another, during which the pulse, while becoming faster, is of very low tension, even dichrotous ("*avec gruppetti*") and this stage corresponds to an acceleration of respiration.

6th minute after blow pulse was	...	68	p.	min.	
7th ,, ,,	...	68	,,		
20th ,, ,,	...	40	,,		
30th ,, ,,	...	54	,,		
2 hours ,, ,,	...	60	,,		
4 ,, ,, ,,	...	93	,,		
8 ,, ,, ,,	...	82	,,		

This observation seems to agree for the most part with what I have gathered from the results of my experiments, which I may here say were not performed to determine this point particularly, and so are less detailed than they would have been otherwise. The conclusion these observations warrants is, that following a severe blow to the head there is usually a certain degree of slowing of the heart, and in all cases a profound disturbance of the general vascular system, a disturbance which must seriously affect the organs of the central nervous system.

Does the Brain Become Anæmic or Hyperæmic?

It is well known from the researches of Vulpian (30), Claude Bernard and others, that a reflex vascular spasm may be produced by irritation of sensory nerves of the body, or by stimulation of the restiform bodies, thus raising the general arterial blood tension, and that this increased tension is due to the contraction of the peripheral vessels

with accumulation of blood in the larger ones, and increased resistance to the heart's action. That such a spasm of the vessels of the brain exists, was demonstrated by Beck, who showed that under such conditions the surface of the brain was pale, and the ordinary pulsations invisible. Duret has confirmed this observation, and adds that the vascular spasm increases in proportion with the amount of violence used. Roy and Sherrington (47), however, state that direct stimulation of the medulla causes congestion of the brain, the vessels passively dilating, but they introduce a fallacy into their experiments by providing for a free escape of the cerebro-spinal fluid, so depriving the vessels of what support they receive from that fluid, and leading to their dilatation.

I hope to be able to show later, that in head injuries there is such an irritation of the restiform bodies as would account for the origin of such a reflex action. Meanwhile let me remark that the result of this alteration in the vascular supply of the brain, whether by spasmodic contraction of the vessels, or by their passive dilatation, will be to deprive the nervous substance, for a time at least, of the copious supply of pure oxygenated blood which is so necessary for the perfect performance of its functions ; in other words it produces a temporary anæmia of the brain, just as would be done by bleeding an animal suddenly and almost completely, or by injecting lycopodium seeds into its vessels.

Kussmaul and Tenner (51) made some experiments on the effects of bleeding animals, and in these they produced symptoms which very closely resemble those observed in a number of my experiments, where very severe blows were dealt to the head, such symptoms as tetanism, marked changes in the pupils, localised convulsions, and so on, being very common.

In support of the contention that in my cases the phenomena were due to cerebral anæmia, just as they were in the animals which were bled, I may quote the conclusion come to by these observers, after they had produced similar symptoms by cutting off the blood supply to the brain. They say " that the interruption of the conveyance of arterial

blood to the brain produces tetanic symptoms as surely as hæmorrhage does."

Sir Astley Cooper (52) demonstrated the same point when he tied both carotids and vertebrals in a dog, with the result that the animal "lost sensation, had great respiratory trouble, with dilated pupils, it fell on its side and became violently convulsed. Stupor and general paralysis lasted for two days, and then gradually passed off, the animal recovering completely."

The series of symptoms produced in this animal, and undoubtedly due to cerebral anæmia, corresponds so closely to what I have again and again seen in my experiments, that I feel considerable confidence in believing, especially when supported by the facts brought out by Vulpian and Bernard, that the pathological condition in both is essentially the same, the only difference being in the originating cause of the anæmia.

To sum up then, I think the weight of evidence is in favour of the view that the symptoms of cerebral concussion are due to a profound disturbance of the circulation of the brain, and that the alteration is in the direction of anæmia.

On the Evidence in favour of Stimulation of the Corpora Restiformes being the cause of the Vascular Disturbance in Cerebral Traumatism.

While making observations on a series of cases in which rabbits, pigs and birds had been subjected to blows on the head, varying in severity and in the exact region to which they were applied, I was struck with the similarity in the symptoms which immediately followed such injuries.

Let us take the case of a rabbit to which a severe blow had been dealt, as an example.

In the first place, the animal deeply anæsthetised, was thrown into a violent tetanic state, with greatly exaggerated motor phenomena, these lasting only a few seconds, and being followed by other motor symptoms less violent and more localised, such indeed as might be attributed to some lesion of a particular part of the brain. These effects I have

observed in a great many of my experiments, in fact, in all in which the blow has been of even moderate severity.

As time wore on the phenomena of this stage, which Duret has appropriately called the "tetanic stage," were gradually replaced by others of a more serious nature. The rigidity and jerkings of the limbs gave place to paralysis, the animal could no longer be roused, and the respiratory and cardiac functions were so interfered with as to cause death.

The symptoms of the tetanic period are illustrated by the following experiments—in all of which, save those on pigs, the animals were fully anæsthetised before the blow was delivered, so that the element of pain does not require to be considered. The pigs suffered from swine fever and were consequently being killed at the Edinburgh slaughter house, where I had the opportunity of observing them.

(1) An occipital blow delivered to a rabbit, was immediately followed by spasmodic convulsions of all the muscles of the body; and these by violent seizures which caused the animal to rotate round its long axis.

(2) A severe blow over the frontal region of a young pig caused the animal to fall. It then lay on its back for a few seconds, all its limbs, but especially the hind ones, being violently moved. There was no distinct opisthotonos.

(3) In another pig a blow of moderate severity was dealt over the vertex. With a scream the animal passed into a state of extreme opisthotonos; for an instant it was in a state of extreme rigidity, and then all its limbs became violently agitated.

(4) In this case the blow was delivered over the left parietal region of a rabbit, while the creature was deeply anæsthetised, and rigidity of all the limbs resulted at once.

(5) In this case the blow was over the occiput, the subject being a young domestic cock, and the result was the same; the creature passing into a condition of extreme tetanus, followed for a few seconds by jerking of the limbs.

These examples will suffice to show that in cases of severe head injury in animals of different genera and even classes, and applied to different parts of the head, practically the same results are produced in the first few seconds after the hurt has been sustained.

And if we look at the gross lesions found in these cases after death, we fail to discover a satisfactory explanation of all the phenomena—opisthotonos, pleurosthotonos, agitation of all the limb muscles, sometimes spasms of the facial muscles, the ocular muscles, and even a combination of all these.

But even did we find a hæmorrhage or a laceration in some particular part of the brain, which could have been the cause of symptoms so serious and so widely diffused, we have still to face the difficulty that these symptoms follow so rapidly on the blow that they cannot be attributed to lesions, which, from their nature must of necessity take some little time to be produced.

It seems therefore that this consideration strengthens the view that the phenomena are due to a reflex action, as was first pointed out by Duret.

But for a reflex action we require an efferent stimulation. What then is the area stimulated in these cases? We know from experience that injuries to the scalp, even much more severe than those produced in the cases quoted, do not give rise to such symptoms; and it has been shown that direct stimulation of the nerves of the dura mater likewise fails to produce such a gross reflex manifestation.

We must therefore look to some part of the brain itself for an explanation, some part which has a particularly sensory function, and in which lesions are constant in such cases.

The notes of my observations shew that the area most frequently injured is the bulb, and it has been pointed out by Duret that the most sensitive part of the bulb is the restiform body, by irritating which *mechanically* he has succeeded in producing all the phenomena of this tetanic stage of concussion. He says that "the phenomena of the first stage of concussion find a very satisfactory explanation chiefly in the irritation of the restiform bodies. . . ." and this statement is supported by the fact that in all the cases just referred to there is evident enough source of irritation of these bodies, and that in those in which the symptoms were most marked the lesions are also most prominent.

The next points to be decided are—"How are these parts stimulated? How are the lesions so often found in the region of the bulb produced?"

On the Anatomy and Physiology of Cerebro-Spinal Fluid.

The physiological importance of the cerebro-spinal fluid has been carefully studied by John Hilton (41), Richet (43), Salathé and others, who have shown that in all probabilty its function is to permit of the variations in volume of the brain which are known to accompany functional activity, great muscular or respiratory exertion and so on.

Now it is evident that the rigid cranium can take no part in providing for these variations in size, and it is equally evident that they must be provided for, because the delicately constructed cerebral tissue would not be tolerant of such pressure as would otherwise inevitably result, without exhibiting important symptoms. We would therefore conclude, and there is abundant experimental and clinical evidence in support of the view, that the cerebro-spinal fluid is the safety valve of the brain.

Believing that the cerebro-spinal fluid is the all-important factor in the production of the phenomena of concussion, and of the lesions found in severe head injuries, I must briefly consider the anatomical distribution of this fluid in the nerve centres.

A. Everywhere the brain is surrounded by a layer of cerebro-spinal fluid, varying in thickness or depth at different places, constituting what has been called the "water bed of the brain." The main "lakes" or "cisterns" are situated in the region of the base and also along the course of the larger blood vessels, such as the sylvian artery.

B. The brain is hollowed out by a series of cavities—the ventricles—and these are filled with cerebro-spinal fluid.

C. In connection with each blood vessel as it enters the brain substance there are two distinct lymph spaces:—(*a*) The adventitial lymph space of Robin, between the tunica muscularis and the tunica adventitia, and continuous with the sub-arachnoid space; and (*b*) the peri-vascular lymph channel of His, between the adventitia and the brain substance, and continuous with the epicerebral space. These spaces, the latter of which continues to surround the blood vessel on to its minutest ramifications are filled with cerebro-spinal fluid, in which the blood vessels are

suspended. This is the anatomical point previously overlooked in this relation.

D. And lastly, the nerve cells themselves are similarly placed, each cell being, as it were, suspended in a small excavation in the brain substance, called a pericellular sac. The communication between the perivascular spaces, and the pericellular sacs depends on the connective tissue cells of the neuroglia, which are distributed along the adventitia of the arterioles, and on to the arterio-venous capillary plexus, and thus establish a connection between the sacs and the spaces.

We are able to trace in successfully prepared sections, in close proximity to every nerve cell a branch of a capillary vessel, and it is to the sides of this loop that the pericellular sac is attached, the nerve cell being apparently suspended in the sac by means of its processes, which, passing through the free space of the sac proceed in all directions into the neuroglia. As these sacs are never terminal the nerve cell is bathed in a constantly renewed lymph bath.

In addition to these perivascular spaces and pericellular sacs, Bevan Lewis (37) describes a terminal set of lymphatic elements which consist "of a system of plasmatic cells with numerous prolongations, which are always in intimate connection with the adventitial lymph space, and which drain the areas between the vascular branches, these we have termed the lymph connective elements."

These four elements, all in direct anatomical continuity with one another, go to make up the cerebro-spinal fluid system of the brain.

Here I may remark that the fluid surrounding the brain is in direct continuity with that around the spinal cord, and that its transit from one cavity to the other is a comparatively easy matter; while the intra-ventricular and sub-arachnoid fluids communicate directly only through a very small aperture in the region of the fourth ventricle—the foramen of Magendie (35).

On Duret's Theory of Cerebral Concussion.

The research which led M. Duret to formulate the cerebro-spinal fluid theory of concussion was carried out in the laboratory of M. Tillaux, and the result published in 1878.

In the first of his experiments he forcibly injected into

the cavity of the cranium of a dog, through a small trephine opening, about 300 grammes of water. After severe spasms of the limbs, opisthotonos, and cardiac and respiratory troubles, the animal speedily died, and the brain presented quite a series of very severe lesions, such as a rupture in the floor of the fourth ventricle, tears in the substance of the pons and medulla, hæmorrhages into the restiform bodies, third ventricle, and on to the surface of the brain.

Believing that this enormous destruction of brain tissue might be due to the increased pressure produced in the cranial cavity, and especially in the ventricles, by the large quantity of fluid so rapidly injected, he varied his next experiment by using a coagulable fluid, namely, gelatine, injecting it very forcibly between the dura and the skull. After exhibiting symptoms similar to the last animal, only somewhat more exaggerated, death followed in a few seconds. The lesions corresponded in a general way to those in the previous case, but were on the whole more severe.

These lesions M. Duret believes are all due to the sudden forcible pressing of the fluid of the lateral ventricles backwards through the aqueduct of Sylvius on to the fourth ventricle, and thence to the cord. The fluid in the lateral ventricles being five or six times more than the fourth ventricle can possibly contain, the latter cavity is subjected to such enormous pressure that its walls are torn.

On comparing these results with my own, produced by the less artificial means of administering blows to the surface of the skull, one cannot but observe the remarkable correspondence in the two series, not only as regards the symptoms shown, but also in the nature and distribution of the lesions observed after death.

It is, however, to be observed that in the injection experiments the symptoms and lesions are alike much more serious than in the percussion series—doubtless on account of the greater force employed, and the more direct way in which it is applied to the brain and its contained fluids.

But, however similar the results in the two cases may be, I cannot admit that the means of producing these are so nearly comparable as M. Duret would appear to indicate. In

the one case the force acting on the head is momentary in its duration, and the cranial contents could, but for any lesions produced by the blow, readily resume their normal conditions and continue to act as before. In the other series, however, the material injected necessarily remains, and so profoundly alters the physical and physiological conditions of the nervous centres that a return to their *status quo*, even supposing no lesion had been produced, would be absolutely impossible. In this, therefore, I think Duret's experiments are much better adapted to the elucidation of the symptoms and pathological appearances of *compression* than of *concussion*. At the same time, they have served to draw attention to the mechanism by which concussion symptoms and lesions—for I think we must admit there are lesions in concussion—are produced, and it is to the confirmation of these observations that I desire to direct my attention, by experiments performed more in accordance with the conditions under which our patients receive their injuries, and therefore more likely to lead to a useful clinical appreciation of the at present obscure symptoms, more obscure pathology, and still more obscure remote prognosis in cases of severe head injuries (48).

It is undoubtedly exceedingly difficult to distinguish clinically between Concussion, Compression and Contusion of the brain, and even after the most careful examination of the symptoms in an individual case, even experienced surgeons are unable to form a decided opinion. This is of course due to the fact that these pathological entities seldom exist singly—a blow which is serious enough to produce marked symptoms of concussion, is usually sufficient to cause gross lesion of the membranes, giving rise to hæmorrhage enough to cause symptoms of compression, which take the place of those of concussion as they tend to pass off. Or should the membranes and their vessels escape laceration, the nervous substance itself suffers injury enough to permit of blood being extravasated, and so raising difficulties in the diagnosis. M. Duret in speaking of this difficulty speaks of "doubt, which has not ceased after the autopsy."

On the Mechanism of Cranial and Cerebral Injuries.

With these preliminary observations we may proceed to consider how severe blows and other injuries to the head produce their effects.

"*Cone of Depression*," and "*Cone of Bulging*."

It has been shown by Félizet (44) that the skull is elastic in a high degree, so much so, that it will rebound from the ground to a considerable extent if dropped from a height.

By smearing the vault of the skull with ink, and then dropping it from a height on to the floor, and comparing the size of the stain left with the area of rounded skull which could be in contact with the flat floor at one moment, he found that the former exceeded the latter in size. This could only be accounted for by the area around the point of contact being momentarily increased by the weight of the skull.

In another series of observations the skull was filled with soft paraffine, and then allowed to fall forcibly on to the ground, and it was found that a large depression was left on the paraffine, exceeding in size the possible area of contact, being distinctly rounded in outline and deeper in the centre than at the margins.

This circle of depression, or as M. Duret more accurately describes it, this "cone of depression," represents the extent to which the cranial parieties are depressed short of being fractured.

In order to account for certain phenomena observed in cerebral traumatisms, Duret states that at the opposite pole of the axis of percussion, the vault of the cranium presents a corresponding "cone of bulging." He does not, however, state whether he merely assumes this cone of bulging, or whether he has demonstrated it. If the latter, he does not describe the method by which he has done so, nor does he quote the experiments of others bearing on the point.

Experiments on Production of Cone of Bulging.

As I believe this to be a most important step in his line of argument, I have made a number of experiments to satisfy myself as to the accuracy of his statement.

These I did on the cadaver as soon after death as I could obtain an opportunity, so that the physical conditions might be as nearly similar to those existing during life as possible.

I obtained a quantity of glazier's putty, of such consistence that it could be handled without losing an impression made on it, and having oiled the skin to prevent too close adhesion, spread a pretty thick layer over one or other frontal region. I then marked out what I judged to be the opposite pole of an axis of percussion, and dealt a sharp blow over that spot with a wooden mallet.

In a majority of the cases a distinct impression was left on the putty. I varied the position in which I dealt the blows, with similar results.

In another set of experiments with the same object, I spread a smooth layer of vaseline about a line thick over the forehead and then dealt a smart blow over the opposite pole.

In some, although not in all cases, a clearly marked ring could be seen on the surface of the vaseline, standing out very much as butter stands out from bread when two surfaces have been separated. In one of the cases in which no such ring could be detected, the head had come off the mallet, and the force thereby misspent; in the other negative cases the only explanations I can suggest are (1) The blow not being strong enough to depress the skull at the point of contact, and (2) the blow being too diffuse to form a defined "cone of bulging."

The results, however, seem to confirm what Duret had assumed (or proved), and are, I think, of importance in so far as they help to explain the mechanism of lesions so often found at the opposite pole of the axis of percussion, in head injuries—that is, at the so-called point of *contre-coup*.

In view of these facts, I take it that the mechanism of

an injury to the head producing gross lesions, is, briefly, this :—

A blow is dealt to the cranial wall, and surrounding the point of impact the skull is depressed, forming the "cone of depression," and from the area into which this cone projects the cerebro-spinal fluid is forcibly expressed. Where does this fluid go? We have seen that under such conditions, at the opposite pole of the axis of percussion, there is formed a "cone of bulging," which would accommodate all or most of this displaced fluid. What does not find accommodation there will either pass along the perivascular lymph spaces into the brain substance, along the nerve sheaths, or into the subarachnoid space of the cord.

The cones of depression and bulging, however, are only of momentary existence, the elasticity of the skull permitting it at once to return to its *status quo*. Therefore, at each point there is formed a vacuum, first at the cone of depression when the bone recoils, and next at the cone of bulging when the fluid recedes. The result of this is, that the blood-vessels of the membranes and brain substance at these points are left for the moment unsupported, and they rupture, a result which is favoured by the fact that the compression to which they have been subjected has partially paralysed them.

For this rupture of vessels suddenly deprived of support derived from fluid under tension we have several surgical analogies—notably where, after withdrawing all the fluid from an over-distended bladder, we have hæmorrhage from the vessels of the lining membrane, or it may be even from the kidney, if the backward pressure has been sufficient to affect that organ.

Such a mechanism would explain the occurrence of lesions, not only at the seat of injury, but at the opposite point of the skull and brain, the so-called lesions of *contre-coup*.

On "Contre-Coup."

That this term *contre-coup* has become a most useful one clinically there is no doubt, but it is by no means so certain

that it has as satisfactory a pathological significance; and it is still more doubtful if those by whom it is used always entertain any very definite idea or theory as to what it means.

Mr. Erichsen (45) says that "the explanation . . . is, that the blow starts a wave in the soft cerebral substance, which breaks against the bone on the other side. In very rare cases it is possible to trace the course of this wave by hæmorrhages in the cerebral substance in its track." We have already seen that Gama (23) and Alquié deny that the brain elements can be thrown into vibrations by a force applied to the surface of the skull.

Le Gros Clark (6) denies the admissibility of the term *contre-coup* in the physics of the cranium at all. He points out with some considerable show of reason, that while a series of vibrations set up at a particular point in the circumference of a perfectly spherical glass globe, might find a point of convergence opposite the point of impact, such a result is physically impossible in the skull. When we consider its irregular shape, its varying thickness, the projections externally and internally, the numerous muscular attachments on its outer aspect, the adhesion of the dura mater and its processes on its inner, and the presence of the cerebro-spinal fluid—all of which would either absorb or transmit in different directions the vibrations set up, we see how exceedingly improbable it is that these vibrations would ever find a point of convergence, and especially one situated exactly at the opposite pole of the axis of percussion.

Although we are not here specially concerned with the question of fracture of the skull from *contre-coup*, it bears so close a relation to what we have just considered, that we may glance at it in passing. That it cannot be due to vibrations finding a focal point at the seat of fracture is, I think, beyond doubt, and it is scarcely more likely that the force of the soft resilient brain impinging on the hard, elastic skull could cause fracture of the latter.

In spite of the strong opinion of Mr. Erichsen, who "cannot doubt it," as he has "seen unequivocal instances," I think Mr. Le Gros Clark is nearer the truth when he says

that "a blow on one side of the head could only produce a fracture on the other side, if the head were knocked against an unyielding object," and in a great majority of cases of severe head injury the patient does fall, usually in the direction of the blow, thus providing an explanation such as Clark suggests.

While I believe that most of the so-called fractures by *contre-coup* are produced in this way, I think that in the light of the experiments on the "cone of bulging," others may be explained on the theory that this cone has gone beyond a point from which it could recoil and the bone has given way. After breaking, the skull might resume its previous level and the only evidence be a fissure; or, as in the rare cases quoted by Mr. Walsham (46), the fractured bone raised above the level of the skull constitutes what is called an "elevated fracture." A specimen of this condition is in the Museum of St. Bartholomew's Hospital, No. 879a.

Fracture of the base by a fall on the vertex is often referred to as an example of genuine fracture by *contre-coup*, but as Le Gros Clark points out, this is really a fracture by direct violence, the weight of the body being transmitted through the spinal column, and impinging on the occipital condyles. This view is borne out by the fact that very often the basal fracture is much greater than that of the vault, being therefore presumably the primary lesion, and in addition the fracture of the vault often appears to be spread from the base.

If we accept this theory of *contre-coup* lesions—namely, that they are due to the displacement of the cerebro-spinal fluid by the cone of depression, and its sudden recession from the cone of bulging leading to rupture of blood vessels, and consequent contusion and laceration of brain substance,—it will be evident that these lesions will vary in position according to the point struck.

If the frontal region or vertex be the seat of the cone of depression, it will be at the base of the skull that we must look for the evidences of the cone of bulging. We can scarcely expect, however, that any ordinary violence will cause an outward bulging of the body of the sphenoid bone,

and we have already seen that when it is fractured it is not from violence applied to the vertex, but to the superincumbent weight of the body. The increased quantity of fluid therefore must swell the volume of the cerebro-spinal lakes at the base, and thus the force will be transmitted to the nerve elements themselves. That all fatal cases of concussion are associated with peribulbar lesions was stated by M. Fano in 1853, and with him in a general way Duret agrees. The results of my own experiments, which will be detailed later, have been to confirm these observations.

The late Mr. Vincent, quoted by Spence (47), evidently appreciated this element of the transmission of blows to the base of the brain, because he states as his opinion, that the amount of danger in cases of concussion depends largely on the direction, as well as the degree of the force, being greatest when in a line directed towards the base of the brain.

Mr. Bryant (48) also, by clinical observations, bears out the opinions of the various experimenters named, when he says that "in almost all cases of severe concussion the base of the brain is injured."

Experimental Evidence in Favour of Cerebro-Spinal Fluid Wave Theory.

After a blow to the head, in addition to the wave set up in the cerebro-spinal fluid external to the brain, *i.e.*, in the sub-arachnoid space, there is another and perhaps a more important disturbance, in the intra-ventricular fluid.

When the cortical substance of the hemispheres is pressed upon, it finds in the ventricles and their contents a sort of buffer which enables it to withstand shocks in a manner which would be impossible were each hemisphere a solid mass of brain substance.

Now, let us suppose the head is the subject of a severe blow, say over the frontal bone, a cone of depression is formed, which displaces the sub-arachnoid fluid in this region, and by the mechanism which we have already considered, a laceration of the brain or its membranes may take place. Not only so, but the force passing through the frontal lobe sets the fluid contained in the lateral ventricles into motion

in the direction of the axis of percussion, *i.e.*, towards the base of the brain. Here, however, there is considerable difficulty, because the only point of escapement is through the narrow aqueduct of Sylvius, and through this the fluid rushes, the effect on the aqueduct and its boundaries varying with the force employed, and its direction. When the force is great, the aqueduct may be torn up and lacerated, while in less severe cases it is only stretched. Whichever condition exists, after travelling the narrow aqueduct the fluid impinges with increased force on the wider fourth ventricle, which, as Duret puts it, "forms a cone of bulging ready prepared," with the result that the pressure on this most important locality is enormously increased, not only by the surplus quantity of fluid thrown in, but by the difficulty with which it can escape, there being only the minute central canal of the cord, and the scarcely larger foramen of Magendie to provide for the overflow. Under such circumstances we are not astonished to find primary lacerations of these delicate structures.

We have seen that the lesions on the surface of the hemispheres are in all probability due rather to the aspiration of the receding cerebro-spinal fluid, than to the pressure of the impinging wave, and I believe it is so too, in the ventricles and at the bulb. It is not uncommon to find, as I shall be able to show from my experiments, that the lateral and fourth ventricles are the seat of extensive extravasations, due, I consider, to the rupture of the unsupported vessels of the choroid plexuses during the ebb of the fluid wave.

In order to determine if the absence of the cerebro-spinal fluid would lead to rupture of these vessels and consequent hæmorrhage, I made several experiments by aspirating it.

These I shall proceed to describe.

Experiment 1, *Rabbit H.—To determine the effect of rapid aspiration of cerebro-spinal fluid on the vessels of the brain substance, especially those of choroid plexuses.*

November 21*st*, 1890, *noon.*—The rabbit, an adult male of average size, and in good condition, having been brought thoroughly under chloroform, an opening was made through the occipito-

atloid ligament, and the finest needle of an aspirator passed in. The trocar was then withdrawn, the cannula connected with the vacuum, and the cerebro-spinal fluid *rapidly* aspirated. Between two and three drachms were removed. During the aspiration the animal once had several slight spasms of the facial and limb muscles. These were so slight that at the time they were regarded as probably simply contractions of the panniculus muscle, and that they were due to reflex irritation of the left restiform body, which might have been irritated by the point of the needle, which was turned to that side after the membrane was perforated, and was felt to touch something. The cannula being withdrawn, the puncture was covered with collodion and wool. Antiseptics were used at the operation.

After aspiration the animal lay on its right side evidently in a perfectly unconscious condition. Pulse feeble and slow, breathing very shallow and rapid. No other sign of life was given. Loud sounds made close to the ears did not startle the creature.

Notes.—This moribund appearance has continued for an hour. General condition now improving. Pulse better, respiration normal. Both pupils dilated equally. The muscles of the right side of the neck are tonically contracted, forcibly bending the head to that side. All the muscles of the limbs seem to share in this tonic rigidity, though to a less extent than those of the neck. On the other hand, the muscles of the trunk appear to be paralysed, or at least in a state of paresis, because the animal is quite unable to move its body from the ground, although it can move about. In doing so, it exhibits an amount of inco-ordination of the limb movements. It now gives evidence of seeing, and of hearing loud sounds. Died soon after.

Autopsy.—As in life head forcibly turned to the right and dorsiflexed.

Brain.—Hemispheres look pink and congested; vessels on left side, both above and below, distinctly more engorged and prominent than those on right side. No hæmorrhage on surface.

Cerebellum congested in same way as cerebrum. Just over the cerebellum, in the space between the posterior ends of the two hemispheres is a small blood clot about size of split pea.

Interior of Brain.—On opening up hemispheres large clot found filling up the left lateral ventricle, and resting on upper aspect of basal ganglia and corpora quadrigemina of that side. The small clot seen above the cerebellum is found to be the termination of this one. Similar clot but much smaller found in right lateral ventricle.

Bulb.—Over left side of medulla in region of superior olive and restiform body is a small laceration, probably caused by the needle in aspirating. Near lower end of medulla, towards middle line and extending to left side is a hæmorrhage into the substance of the part. It extends along the medulla for about one-eighth of an inch, is irregularly rounded in shape, and has a piece of apparently normal brain tissue in the centre.

The aqueduct of Sylvius is dilated and exhibits one or two small hæmorrhages round it, the largest being in the left side of the corpora quadrigemina.

Cord is very pale and anæmic.

Remarks.— This experiment appears to bear out the theory that the sudden withdrawal of the cerebro-spinal fluid, and the consequent leaving of the vessels unsupported, leads to rupture of the latter and extravasation of blood into the brain substance (*e.g.*, the medullary hæmorrhage) and into the ventricular cavities. It also shows that the rushing of fluid through the aqueduct of Sylvius not only leads to dilatation of that passage but also to hæmorrhage into the comparatively dense tissues which surround it. Of course it may be suggested that the hæmorrhages were due to the aspirating force acting on the blood vessels after all the fluid had been withdrawn. I tried to guard against this fallacy by limiting the power of the vacuum, only partially exhausting the bottle. Besides, one's clinical experience in the use of the aspirator does not give much support to this view.

The symptoms due to the actual withdrawal of the fluid were neither numerous nor important, the animal of course being deeply anæsthetised. I have already said that the slight spasms, if such they could be called, were probably reflex.

The coma, rigidity of the limbs, with deviation of the head to one side, and slight variation in these irritative indications, are to be accounted for by the hæmorrhage into the left lateral ventricle, though why this hæmorrhage should have been more abundant on one side is not so easy to understand, unless, indeed, it be that the turning of the needle point to that side determined the direction of the flow. This we would not expect, because the needle should

have been in the inferior cerebellar lake, and the fluid to reach it would have to traverse the foramen of Magendie, where any inequality in the streams from the right and left ventricles would be equalised. The fluid of course may have escaped from the lateral opening of Key and Retzius. However, working in such a limited space and with such small quantities of fluid, the sources of error are so numerous, that we must not draw too wide conclusions from a single case.

To the hæmorrhages in the corpora quadrigemina we may assign but few symptoms. Abnormalties in the action of the ocular muscles often depend on lesions of these bodies, but I think the general comatose condition of the animal is amply sufficient to account for the phenomena in this case.

The peculiar inco-ordination of movement is explicable on one of two theories. We know that in certain cases of tumour of these bodies this is a prominent symptom, but Dr. Gowers (49) has pointed out that it is probably due to the pressure on the middle lobe of the cerebellum, rather than to any influence on the corpora quadrigemina. Here we had both hæmorrhage into the corpora quadrigemina and pressure, though slight, on the middle lobe of the cerebellum by the posterior end of the intra-ventricular clot. Probably both played a part.

The condition of the pulse and respiration most likely depended on the large hæmorrhage into the substance of the medulla, caused I believe by the recession of the fluid supporting the blood vessels.

The excessive congestion of the vessels of the left hemisphere, doubtless was due to the increased difficulty in the return of the venous blood, on account of the large clot blocking the vessels of the choroid plexus, and secondarily all the vascular arrangements of that side.

So far as it goes, I think this experiment is satisfactory in showing that by rapidly withdrawing the cerebro-spinal fluid, hæmorrhage can be produced in the cavities and substance of the brain.

The next points to determine are, what is the effect of

slow aspiration of the fluid; and what is the result of a severe blow on the head when the cavity has been deprived of its cerebro-spinal fluid.

The following experiment fortunately, throws light on both of these points.

Experiment 2, *Rabbit I.—To determine the rôle of the cerebro-spinal fluid in the production of lesions from external injuries to the head.*

November 21*st*, 1890, 1 p.m.—The subject of the experiment was an adult male Belgian hare, well fed and not very timid. While deeply under chloroform, an opening was made in the skull by means of a small trephine over the left prefrontal lobe, and through this the cerebro-spinal fluid was slowly aspirated, to prevent a too sudden withdrawal leading to rupture of the vessels, as occurred in Rabbit H. Only about two drams of fluid were obtained and this was slightly blood stained.

During the withdrawal the animal spasmodically jerked its limbs, in the same way as, but to a less extent than the animal last operated on.

While still under the influence of chloroform, a severe blow was dealt over the frontal region in a direction downwards and backwards, in a direct line towards the base of the skull.

The animal was thrown into a state of extreme opisthotonos with tonic contractions of all the limbs, lasting for a few seconds. This passed off, leaving only a distinct paralysis of the right fore limb. It was lying on its side. Some slight symptoms of compression gradually supervened but did not become serious. But for some drowsiness and deafness the animal seemed to recover completely, and was ultimately killed by chloroform inhalation.

Autopsy.—Scalp.—The incision in the scalp had healed by first intention. There was no ecchymosis into the scalp in the region of the blow. *Skull.*—The trephine opening was found to be over the left frontal lobe about the level of the anterior edge of the orbit. It was filled by a small mass of purulent looking material. No fracture of vault, and no contusion of pericranium. At the base there was a transverse fracture right through the body of the sphenoid bone, which permitted a bristle to be easily passed through into the pharynx.

Membranes.—Between the dura and the bone on the right side, there was a thin clot about the size of a split pea, situated on the upper aspect of the orbital plate. This clot was partly decolorised.

Hemispheres.—Just beneath the seat of the trephine opening, *i.e.*, over the left olfactory lobe, and inside the dura was a small clot, presumably due to the needle having injured the brain. The whole upper aspect was normal, and the lower paler than normal. No evidence whatever of blood clot either old or recent.

Cerebellum.—Normal.

Interior of Brain and Ventricles.—Normal. In the aqueduct of Sylvius was what appeared to be a small decolorised clot.

Bulb and Spinal Cord.—In the upper part of the spinal canal there had been some effusion outside the membranes, but it had been small in qnantity, and by the time of death was nearly all absorbed.

Eyes.—There was no extravasation into the sheath of either optic nerve, and no subconjunctival ecchymosis.

Ears.—Right: Large red clot filling up middle ear. Membrane ruptured. *Left:* Partly decolorised clot in middle ear. Canals normal. Membrane ruptured.

Remarks.—On comparing this experiment with the last, we see that although in both the cerebro-spinal fluid was aspirated, the results differ considerably, and I attribute this to the varying rapidity with which the fluid was withdrawn. In rabbit H the fluid supporting the blood-vessels was rapidly withdrawn, while in this case it was slowly removed by gentle aspiration, thus giving the vessels time to adapt themselves so as to compensate for their loss of support. This, I take it, bears out the theory that in concussion the hæmorrhages found in such widely separated parts of the brain are due to this rupture of vessels suddenly deprived of the support of the cerebro-spinal fluid.

That the rôle of the cerebro-spinal fluid is an all-important one in concussion is further borne out by the result of the blow given to the head after removal of the fluid. If we look at cases of rabbits, pigs and birds, in every way identical, save in this one particular, that the fluid has been withdrawn, we find that while in them abundant lesions in the shape of hæmorrhages on the surface of the hemispheres, bulb and cord, into the substance of the parts, and into the ventricles occur, it is only very rarely that we have fracture of the strong base of the skull. Here, however, the conditions are entirely

reversed, because while we can only find two hæmorrhages on the surface attributable to the blow, and both of these external to the dura, and the ear lesions, there is a complete fracture of the basi-sphenoid. Let me point out that this process lies directly in the line of the axis of percussion, and would therefore be at the seat of the "cone of bulging." The cerebro-spinal fluid not being available to protect it, the force has spent itself on the bone.

In spite of the absence of the fluid however, there were some hæmorrhages resulting from the blow, *e.g.*, hæmorrhages into both middle ears, with none into internal auditory meatus. These can be explained on the theory that they were secondary to the fracture of the base. It is to be noted that the other extravasations are all outside the dura, and therefore more likely to be due to fracture than to cerebro-spinal disturbance. The bleeding from the mouth and nose also were doubtless secondary to the basal fracture.

The evidences of deafness exhibited all along, are of course quite intelligible in the light of the *post-mortem* appearances.

The absence of extravasation into the sheaths of the optic and auditory nerves, is due to the want of the cerebro-spinal fluid. At the moment of percussion these perineural spaces were not dilated, and so their vessels were not put into the dangerous condition which leads to rupture.

As a contrast experiment to the above, let us look at that performed on Rabbit C. In this experiment the blow was administered on the occipital region, but in every other respect, save that the cerebro-spinal fluid was not withdrawn, the two cases are similar.

Experiment 3, *Rabbit C.—To compare results of severe blow to head with cerebro-spinal fluid in situ, with those of previous experiments where it had been aspirated.*

November 30th, 1890, 10.45 a.m.—The subject was an adult grey rabbit, not in very good condition, but quite active.

Before any blow was administered the animal sat quietly, its pupils were equal, pulse small, rapid, 180; respiration also rapid, 90.

Anæsthesia having been induced by chloroform, a severe blow, intended to be lethal, was dealt over the middle line in the

occipital region, with the knob of a Kaffir stick. Immediately the creature fell on its right side, all the limbs were spasmodically convulsed for a few seconds. The animal then rolled over and over around its long axis five or six times, and came to rest on its left side. While in this position all the limbs were making violent movements similar in character to those made by an animal galloping, only very much exaggerated. For the first few seconds the fore paws were rigidly extended, but they soon took on the same actions as the hind ones. The head was dorsiflexed, and the tail extended in a straight line with the body. The pupils were equally contracted. This condition lasted for two minutes, then the limbs assumed different positions; front limbs were rigidly extended, and if flexed passively they immediately assumed their former position. The right hind limb was in the same attitude, the rigidity, however, being less marked, while the left hind leg was flexed; the head remained dorsiflexed; pulse full, bounding 120; respiration 124, shallow, very irregular and intermittent, mainly costal and noisy, whistling through the nares.

The pupils subsequently dilated equally, and the limbs exhibited regular and rythmical movements as of walking. The heart became weaker, and the respiration more difficult, and the animal was killed by chloroform inhalation.

Autopsy.—*Scalp:* Large hæmatoma over occiput and extending down as far as level of third cervical vertebra. *Skull:* Depressed and comminuted fracture of the right occipital bone; no basal fracture.

Membranes.—Only slight hæmorrhage from fracture between dura and bone.

Hemispheres.—Under the dura over the left cerebral lobe is a large extravasation, consisting of several large clots with thinner clots between; a smaller clot over the right lobe.

Cerebellum.—Over the middle lobe of the cerebellum is a blood clot of considerable thickness. It extends on to both lateral lobes, and is found to be continuous with an intraventricular extravasation described below.

Interior of Hemispheres and Ventricles.—The left lateral ventricle is filled completely by a large clot which is found to be continuous with that lying on the posterior aspect of the occipital lobe, and on the cerebellum.

This same clot extends into the third ventricle. A clot of considerable size fills the cavity of the fourth ventricle, and on being removed exposes several smaller extravasations into the

substance of the floor of that cavity. The part of the cerebellum forming the roof of the fourth ventricle is pulpy and infiltrated with blood.

The Sylvian aqueduct is enormously dilated, almost sufficient to admit a toothpick. It is filled with clot. There is a small hæmorrhage into the substance of the anterior corpus quadrigeminum of left side.

No hæmorrhage into basal ganglia.

Bulb and Cord.—Various hæmorrhages on base of brain. There is a tear in the substance of the medulla on right side, but no visible hæmorrhages into substance beyond those mentioned. Some extravasation into upper part of spinal cord.

Eyes.—Well marked extravasation of blood into optic nerve sheaths of both eyes, extending backwards from the eyeballs for about half an inch.

Ears.—Right: Some extravasation into the first part of the auditory nerve sheath, in the internal auditory meatus. Canals and internal ear normal. *Left:* Numerous well marked points of ecchymosis into the membranous semi-circular canals, and also hæmorrhages into the middle and internal ear.

REMARKS.—We may compare in a general way the two experiments on rabbits I and C. In the former the cerebro-spinal fluid was aspirated before the blow was dealt, in the latter it was left, and the result of that blow is of the greatest possible interest, because, while in rabbit I the brain tissue practically escaped serious harm, in rabbit C the gross lesions found in the substance of the brain were so numerous, and so widely spread as to be spoken of without exaggeration as universal. On the other hand, in the case where the fluid was absent the base of the skull was fractured, while in the other it was uninjured. When we consider that the intensity of the blow in these two cases was as near as may be equal, the results of the experiments lend great support to the cerebro-spinal fluid theory of cerebral trauma.

If we now compare the result of experiment on rabbit C with that on rabbit H, in which the cerebro-spinal fluid was rapidly withdrawn, we find that in the two the symptoms during life and the *post-mortem* appearances, so closely correspond, that we must consider that in both, the lesions are produced by the same mechanism, a mechanism differ-

ing in the two cases only in the means by which it is started. With so many gross lesions any one of which might produce symptoms, it is useless to attempt either to assign a symptom to each lesion or to each symptom a lesion. Rather must we look upon the injuries to the brain tissue as an index of the severity of the blow, and not as the definable causes of the phenomena.

The presence of blood extravasations into (*a*) the sheath of the optic nerves; (*b*) the sheath of the auditory nerves; (*c*) the aqueduct of Sylvius; (*d*) the lateral, third and fourth ventricles, is of importance for my argument as showing the role of the cerebro-spinal fluid in the production of lesions in cases of concussion.

I have thus shown by experiment that sudden aspiration of cerebro-spinal fluid leads to extravasation of blood; that slow aspiration does not do so; that a blow on the skull after the fluid has been so removed causes much less destructive lesions than when that fluid has been left in its normal position.

The rôle of Cerebro-spinal Fluid in producing Hæmorrhage into Nerve Sheaths.

I have noticed in several cases that, as a result of severe blows, there were found extravasations of blood into the sheaths of different cranial nerves, and this fact I think throws considerable light upon the mechanism of head injuries. It is an anatomical fact of some importance that the cranial nerves have a reflection of the arachnoid membrane accompanying them as far as their foramina of exit from the skull.

Axel Key and Retzius (32) have shown by their injection experiments that the coloured liquid can be made to penetrate into the lymphatic channels which Ranvier (50) has shewn to exist around the fasciculi of nerves, and Schwalbe (33) has demonstrated that these intra-nervous channels communicate with serous or lymphatic spaces of the organs of sense. He states that, "these intra-nervous, serous canals communicate with certain spaces in the organs of

special sense, which we have described by the name of serous spaces or lymphatics. Besides, by the internal auditory canal the serous canals of the auditory nerve are in communication with the spaces filled with peri-lymph contained between the osseous and membranous labyrinth; by the optic foramen the serous spaces of the lamina fusca which accompany the vessels of the choroid, and communicate with the anterior chamber of the eye, are in relation with the serous canals of the optic nerve, and with the central lake at the level of the chiasma; an analogous arrangement exists in connection with the olfactory nerves."

In this relation also, Duret announces this general law, that "wherever we have a nervous element and a blood vessel nourishing it, around the vessel is disposed a serous space destined to receive in the case of excessive tension in the latter the aqueous part of the blood which it contains."

This being so, it is evident that the wave set up in the cerebro-spinal fluid by a blow impinging on the skull, will be transmitted along these peri-vascular channels, and cause distension of them, which will result in rupture and extravasation when the fluid recedes.

It appeared to me that if this fluid could escape from the nerve sheath, and thus obviate its distension, such hæmorrhages would not occur, and to test this point I performed experiment 4.

Experiment 4, *Rabbit F.—To determine the rôle of cerebro-spinal fluid in producing hæmorrhage into nerve-sheaths, especially those of optic nerves; and other points.*

Subject, an adult male Belgian hare in good condition. Both pupils equal and of medium size before experiment. The animal was put fully under the influence of chloroform, and then the left eye was enucleated, the optic nerve being cut as cleanly, and as far from the eyeball as possible with curved scissors. The orbit was washed clear of all blood, and hæmorrhage stopped by plugging. The animal, still deeply anæsthetised, a severe blow intended to be lethal was delivered on the occiput, having a direction from behind forwards, as nearly as possible in the axis of the optic nerves.

Death was almost instantaneous, no other movement than a

slight twitching of the right hind leg taking place after the blow. The right pupil was much contracted after the injury was inflicted.

Autopsy.—Scalp: Slight ecchymosis under the skin over the occipital bone in middle line. No wound of scalp. *Cranium:* No fracture of vault or base.

Membranes.—No hæmorrhage external to dura. Under the dura over the right cerebral lobe was an extensive but very thin clot. None on left side.

Hemispheres. — Extensive hæmorrhages into the lateral ventricles on both sides.

Cerebellum.—Much injured, covered with blood clot, and on right side more or less pulpy, blood clot and brain tissue being mixed up.

Bulb.—Laceration of wall, and extravasation of blood around the aqueduct of Sylvius, and into substance of corpora quadrigemina. Large blood clot on front of pons and medulla pressing on the superficial origins of the vagi, spinal accessory, and hypoglossal nerves. The floor of fourth ventricle exhibited numerous small petechial hæmorrhages, scattered over it in all its areas. The left restiform body was covered by a large thick blood clot; a smaller one existed further down on same side.

Cord.—Extensive hæmorrhage extended down the cord as far as the upper lumbar region, especially on left side, but also marked on right.

Eyes.—Right: Pupil normal after death. There was a considerable extravasation of blood into the back of right orbit, a clot about size of a threepenny piece pressing on the branches of third nerve. Sheath of optic nerve filled with blood clot. *Left:* The optic nerve had been severed about one-eighth of an inch from the eyeball, and there was no blood around the stump. There was no hæmorrhage along its sheath whatever.

Ears.—No hæmorrhage or escape of cerebro-spinal fluid from either ear. Both membranes intact. *Right:* Some blood in the sheath of the nerve. A few spots in the semicircular canals. None in cochlea or middle ear. *Left:* No hæmorrhage along course of auditory nerve in internal meatus. Semicircular canals filled by a large extravasation.

Jaws.—Right: At the inferior dental foramen by which the branch of the third division of the fifth nerve enters the bone was a fairly large clot of blood in the sheath of the nerve. *Left:* One small hæmorrhage close to foramen, and another, a short distance higher up the nerve.

Remarks.—There can be little doubt that the theory enunciated as to the origin of these hæmorrhages into the sheath of the optic nerve, was the correct one, because in the left eye the conditions were such that the cerebro-spinal fluid rushing along the sheath could escape without dilating it, and no hæmorrhage resulted; while on the right side the distension was followed by a copious hæmorrhage.

Not only in the sheaths of the optic nerves have we this recession of cerebro-spinal fluid, but also in the auditory nerves, fifth nerves, and probably in all the others the same thing happens, as is evidenced by the hæmorrhages found in these various positions.

Referring to the extravasations into the internal ears and along the course of the auditory nerves, one can easily understand how, if the tympanic membranes had been ruptured or previously perforated, a symptom supposed to be characteristic and diagnostic of fracture of the base of the skull, namely bleeding from the ears and "welling" of cerebro-spinal fluid, might have been present while no such fracture existed.

The lesions of the left side of the brain are much less marked than those of the right side, and it appears to me not improbable that this is due to a certain amount of the fluid finding a point of escape through the severed optic nerve sheath of that side, so diminishing the size of the wave set up by the blow.

Summary.

Such are the grounds on which I am led to support and confirm this theory of the important part played by the cerebro-spinal fluid in the mechanism of cerebral concussion and other forms of cerebral trauma.

The conclusions I have formed are briefly these:—

(1) That the group of phenomena, commonly spoken of as "concussion of the brain," is the result of a temporary anæmia of that organ.

(2) That this anæmia is the reflex result of stimulation of the restiform bodies, and perhaps other important centres in the region of the bulb.

(3) That these parts are stimulated by the wave of cerebro-spinal fluid, which rushes through the aqueduct of Sylvius, the foramen of Magendie, and from the subarachnoid space of the brain to that of the cord when a severe blow is dealt over the skull.

(4) That in accordance with the laws of hydrostatics this cerebro-spinal fluid wave will disturb the equilibrium of the ultimate nerve cells throughout the central nervous system.

(5) That the hæmorrhages found throughout the brain substance and on its surface are to be ascribed to the recession of the cerebro-spinal fluid, which naturally supports the blood vessels of the cerebrum.

(6) That the petechial hæmorrhages found in cases of so-called concussion are not the proximate cause of the symptoms of that condition. They are rather to be looked upon as an index of the force producing the injury, than as the cause of the resulting phenomena.

In another paper (48) I have described the microscopic appearances found in these and other cases of cerebral trauma, most of which, I feel sure, may be satisfactorily explained on the same theory as are the grosser lesions to which I have alone referred in the foregoing paper.

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THE ROLANDIC AREA CORTEX.[1]

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SINCE the epoch-making "Researches into the Physiology of the Brain" of Hitzig and Fritsch, and the later labours of Ferrier and the numerous physiologists who have followed, it has been accepted as firmly established that the grey matter of the brain—the cortex—is not only excitable by electricity, but that well-defined centres of motor action are contained in certain definite areas of the convolutions chiefly around the Rolandic fissure, or its analogue in the brains of lower animals. I have from time to time since 1873, endeavoured to show that the proofs brought forward were not sufficient; and that the facts observed by the different experimenters could be explained more satisfactorily as purely physical not physiological, phenomena.

(1) It has not been demonstrated that any other agent but electricity does produce any effect in the motor apparatus of animals when applied to the cortex; and this agent cannot have a well limited field of action, since it diffuses through lines of least resistance. Physical, chemical, or mechanical agents fail entirely to give rise to motor action when used in the same manner as electricity, while it is well known that these last agents all affect nerve fibres. The fact that when a current of minimum strength applied to certain spots in the cortex of the brain of certain animals (species) the experimenter is able to predict the effect to follow, only goes to show that certain points of the grey matter of the convolutions are situated in the line of the least resis-

[1] Paper read before the Neurological Society of London.

tance. In reality, all brains of the same species are furrowed by sulci and offer the same mode of arrangement; as these contain elements which constitute good channels of conduction, it happens that on brains in which such similar circumstances obtain, identical results must follow. Moreover, the pia mater over the Rolandic convolutions is almost a complete network[1] of vaso-motor fibres and cells, and blood-vessels, which penetrate into the convolutions. One system of blood-vessels only ramifies and ends in the cortex proper, the other enters the white substance by means of larger vessels accompanied by nerves and ganglion cells. It follows from this arrangement that the cortex proper is much more vascular and the white substance or fibres a great deal less so; and moreover, that whilst the vessels irrigating the cortex are spread well over, those going into the white substance are isolated from one another. Now I have shown long ago[2] that those points, which when "excited" by electricity give rise to a motor action, coincide with spots where arteries with nerves penetrate into the white matter or strands of fibres. The best method to demonstrate this fact consists in injecting one carotid artery after having marked with ink the position of the "motor centres," ascertained beforehand by means of electricity.

Experiments, well known to all, are appealed to not only as giving a complete demonstration that the cortex is excitable (around the fissure of Rolando), but also as proving the impossibility of the explanation I have given. Putnam made this one experiment: after having found the minimum current sufficient to excite motor action when applied to a certain group of "motor centres," he slices off the cortex, leaving it *in situ*, and then, on repeating his excitation, he finds that the same current is insufficient to call forth motor action. Now this experiment is anything but conclusive, vary it as much as one wishes. First, the physical conditions of the two experiments being different, we can by no means obtain identical results:

[1] Hènocque *Thèse inaugurale. Des terminaisons nerveuses.* Duret, *Circulation de l'Encéphale*, 1874.

[2] Dupuy. *C. R. Soc. de Biologie*, 1887.

indeed, the slicing off of the cortex and the consequent clotting of blood, and the creation of two moist surfaces, evidently alter the distribution of the current; it spreads more, and must therefore be strengthened to give the same results as before. Secondly, I have shown that if the cortex being sliced off and the same minimum current used to ascertain the production of motor action be applied not directly, but after a short time, to the cut surface of the fibres, results are obtained identical with those in the first experiments, when using the same current as applied to the cortex proper before the slicing off; this indeed was to be expected, it proves that fibres below the cortex are excitable, it neither proves nor disproves an excitability of grey matter proper.

Another experiment devised by Frank and Pitres consists in marking the time elapsing between the moment of application of a minimum current to a "motor centre" and the occurrence of motor action; and they found that there is a difference in *plus* equivalent to about one six-hundredth of a second upon the same time elapsing from the moment of application of the same current to the cut surface of white matter (the cortex of which has been sliced off) and motor action following. This longer time they ascribe to the elaboration of nerve force by the cells or reaction time. Bubnoff and Heidenhain and others have confirmed these results. The two last named observers have moreover discovered this remarkable fact, that slight irritation of the skin of the limb of which the "motor centre" is being experimented upon, increases the excitability of this centre when the animal is under the effects of anæsthesia and morphinisation; but from what I have already said, it appears clear that the reaction time of Frank and Pitres is owing entirely to diffusion of electricity in a tissue much more vascular than the white substance fibres under the convolution, as the cortex proper is known to be; and that there takes place a series of actions almost inevitable (electrolysis, short circuiting, &c.), hence the difference in the results. As for the explanation of the fact discovered by Bubnoff and Heidenhain, I find that when they excite

the sensitive nerves of a limb (skin) they certainly bring on vaso-motor change in the brain surface, and since anæsthesia and morphinisation induce contraction of blood-vessels, therefore less nutrition and excitability of nerve tissue, the irritation of the skin of the limb changes that state by *plus*, as I have ascertained in the pupil of the eye which shows the characteristic reaction, known since the early days of Vulpian and Schiff. The facts recorded so far appear to me to allow more than strong doubts being entertained as to the excitability of the cortex proper by electricity, it being inexcitable by any other means. The experiments devised by Frank[1] and consisting in circumvallation of a given centre, go a long way to support my theory. Frank says that: "Having laid bare the right hemisphere of a dog, it is noticed that the animal, after the sigmoid gyrus has been uncovered by section of the dura, behaves exactly like a well animal; the right hind leg appears a little more outward than its fellow. The animal attempts to get rid of the muzzle by means of its left fore paw chiefly. The motor centre for this limb having been ascertained by electricity, a bistouri is passed around the motor area to the white substance, describing a circle around the gyrus, the depth of the incision notably exceeding the depth of the grey matter; *care has been taken to save two bridges of pia mater containing large arterial and veinous vessels.*"[2] The animal left free behaves as before and uses his two fore limbs to get rid of the muzzle, *but just as before the "circumvallation" of the motor centre of this limb, it is the left fore paw which he uses chiefly.* A weak stimulation of that motor centre which is now isolated from the rest of the cortex by the circular incision, at first gives rise to localised motor action in the left fore paw, and then an attack of convulsions which become general, invading all the limbs and lasting a long time, etc.

After renewed weak electric excitations, which show the persistency of the excitability of the circumscribed area of

[1] Frank. "Fonctions motrices du cerveau," p. 459. Marique. *Thèse.*

[2] No italics in text.

cortex, the whole of the grey matter of this area is sliced off, the white surface thus produced being dried up, it is found that it requires a much stronger current to induce feeble motor action in the left anterior paw; the animal falls over the left side and does not succeed in getting upon its legs when left on the floor. In a second animal the same experiment is performed, but in order to avoid flow of blood which had proved embarrassing, the circumvallation of "motor centre" was made with a galvanic cautery (the centre for the left anterior leg) and in such a manner as *to save the large veins;* the section is completed with a bistouri. When left on the floor the animal shows manifest motor troubles on the left side, chiefly the fore left leg. Electrical irritation of that area thus isolated fails to give rise to motor action even with currents progressively strengthened. After removal of the cortex it is found that the white matter is only excitable much deeper, &c. A third experiment is made upon another animal, and the whole sigmoid gyrus on the right side is circumvallated by a bistouri section, but continuity of the superior portion with the rest of the brain is preserved. No trouble of motion whatsoever is observed. The section is completed so as to form an island of the gyrus, and the animal now shows manifest signs of motor troubles in the left limbs, &c., and excitation of the circumscribed area brings forth evident motor action in the left anterior leg (crossed action). One hour later, the motor troubles had greatly diminished, the gait being less awkward, &c.

It appears to me, from these results, that the theory I have advanced (the action of electricity upon white matter through blood-vessels and nerves accompanying) is well supported, and the cause why in one experiment (No. 2) it was necessary to irritate a deeper layer of white matter is only due to the fact that the upper layers of white substance receive their blood-vessels from the cortex, and the lower by the opto-striated system of vessels; and the galvano-cautery action was more than enough to account for the shock effect produced on the white substance, besides coagulation inevitable when cautery is used. I must here state that in

dogs at least the blood supply of the convolutions appear to be less differentiated than in man, for it is impossible not to fill all the arteries of one hemisphere (convolutional chiefly), when colouring matter is pushed through either carotid, all the arteries seem to communicate in the cortex. Frank has criticised my views on the role played by penetrating arteries and nerves in the effect of exciting the strands of white fibres when electricity is applied to "motor centres." If he had endeavoured to understand my view he would have seen that my meaning is simply that these elements offer lines of least resistance; and now that we possess the results of the researches of Golgi and his school, it is quite easy to understand that if the blood-vessels with which the protoplasmic prolongation of cells are in contact is undergoing alteration, the nerve elements will also undergo change. And in last resort we have to deal here with a simple case of animal electricity, positive or negative variation as the case may be, and induced by change in blood supply. When Kussmaul and Tenner, having tied the four blood-vessels going to brain, find that convulsions supervene, they have produced exactly the same change which I claim we produce when we use electricity on 'motor centres,' *i.e.*, electrical variation. I simply stated that the current (minimum) only acted on the "motor point," which is also the point of penetration of the arteries accompanied by nerves; I never stated that the current went all along the vascular supply and selected one artery and its nerves, and therefore I see no reason to ascribe to me the inane theory which Frank has criticised. I have preferred to give as illustration of my views by means of experiments performed by Frank himself, although it is needless to say, he did not insert the record of these for my purpose.

Another experiment of Frank's consists in freezing the "motor centre" and then irritating the hard frozen surface; it is found that whereas before the freezing the motor action in the limb was longer in duration than the current used and was epileptic, and besides only occurred after a lapse of about $6\frac{1}{2}$-hundredths of a second, when the same current is applied during the frozen state the time of reaction is only

about 4½-hundredths of a second, and the contraction is only tonic. The claim is made from these results that the reaction time is greater (6½-hundredths of a second) when the cortex is not frozen because it reacts *physiologically*, and the time is shorter (4½-hundredths of a second) when the cortex is frozen because it reacts *physically*.

This I consider a very unacceptable interpretation; it is unnecessary to state that the white substance as well as the cortex is influenced by the freezing process and the irritability of both endangered and altered; besides, what of the inhibitory action of such a frozen "wedge" into the centrum ovale?

But I have shown that if two fine needles, insulated by varnish altogether and the points only shining, are used instead of ordinary electrodes, and these fixed in a cork and protruding one millimetre and a-half apart, and passed with care below the pia mater (in same manner that hypodermic needles are made to travel into the skin) the reaction time is identical in both cases, whether the electrical minimum currents be passed between the cortex and the pia or just below the cortex.

The figures given by Frank in his book[1] do not support his own theory. In the first graphic the tracing shows that after cortical freezing the contraction graphic line does not last much beyond the time of irritation, and yet the character of the contraction is epileptic. If the cortex reacted physically when frozen the contraction ought to have been purely tonic and to have lasted only as long as the current used, and not to have shown the character of tonic and clonic convulsion. Later, while the cortex was getting unfrozen, and still later when it was unfrozen, the tracings show an increase only in the duration of the epileptic contractions, a result which it is natural to expect, considering the state of change induced in the 'motor centre' area by the preceding freezing—it is inflamed. I much prefer to make use of facts recorded by those authors who hold the doctrines which I consider erroneous to show

[1] Frank. "Fonctions Motrices du cerveau," p. 113. The three graphics are very interesting.

how misinterpretation has led them to frame the doctrine of localisation of motor functions in the cortex. I call such facts "impartial."

Thus the statement that muscle contraction differs when the cortex is excited by being of longer duration than when the white fibres alone are excited, is proved to be erroneous by Frank himself in the experiments which I have reported above, and the value of the distinction in the nature of the muscle contraction, if it did really exist, would be negatived by the fact that complete epileptic convulsions occur even after the destruction of the cortex over the motor area as well as over the whole brain.[1] So far from denying I was the first to assert that soon after slicing off the cortex, electrical irritation of the artificial surface of white substance may give rise to a motor action identical with the one following electrical excitation of the cortex; indeed, nutrition of the elements is highly impaired and altered, and we must take into account the inhibitory action also arising from traumatism, but it remains constant that in this condition the limb, the so-called 'motor centre' of which has been sliced off, becomes the seat of epileptoid contractions, tonic and clonic. Ferrier[2] says that I have degraded the cells of the cortex of the 'motor centres' by stating that they are not the elements upon which electrical currents take effect. I deplore that I am guilty of that great offence; I shall however try to atone in some manner later. I do not believe that anybody can show that grey matter in the spinal cord can be excited into activity by any agent applied directly to it (not through nerves), and the grey matter of the cortex being not of a different nature from that of the spinal cord, there is no *a priori* reason to

[1] After having ascertained 'the reaction time' when the cortex is irritated by electricity, and also when the white substance uncovered by slicing off the cortex, a thin piece of amadou is placed over the white substance in place of the cortex—amadou soaked with cerebro-spinal fluid or physiological salt solution, and the 'reaction time' when electricity is applied to this artificial cortex is apparently the same as when electricity is applied to the natural cortex. It would be rather absurd to speak of amadou reaction time I imagine, although in the case of amadou, as in the case of the cortex, the reaction time is apparently the same, and the muscular response is alike in both instances, being epileptic (tonic and clonic).

[2] Ferrier. "Localisation of Cerebral Disease," p. 16.

suppose that it should be endowed with different properties. The cells of the cortex may well remain undegraded, even if we consider them as Golgi has been teaching, and with him all his school and chiefly Nansen,[1] to be trophic elements; or if we attribute to them different functions than those ascribed to them by Ferrier. There are besides, as I will show in a moment, so many reasons to reject the theory that the cells are either motor or sensory, and that there are in certain areas of the cortex 'motor' or 'sensory' centres, that this doctrine is no more tenable. Already great differences exist between those who teach the doctrine of 'localisation of functions' on the interpretation of the nature of the very function of the cortex. It is not necessary to mention before this Association the hypotheses of Hitzig, and of Nothnagel, who consider the Rolandic region as the centre of muscular sense or muscular consciousness; of Bastian, who separates muscular sense from the other compounds which go to form his 'kinæsthesis'; of Schiff, who believes that it is some kind of sensitive region in connection with tactile sensation, said by him to travel through the posterior spinal columns; nor the doctrine of Ferrier, who considers the Rolandic region as entirely motor—a doctrine which is identical with the one taught by Hughlings Jackson (the original founder of the doctrine); nor of Charcot, who holds that the "motor centres" are the substrata of motor action as distinguished from sensation and sensory centres; nor Munk's psychic theory of sensory spheres representing images of the different species of sensations common and special, and which react in a psychic manner —"metaphysiological," if I may so express it. There are also a number of theories, chiefly evolved by the Italians, which are modifications of those given above. And I have only thus briefly enumerated them to give prominence to the want of agreement as to the specific nature of the 'functions' of the cortex of the Rolandic region.

But a more practical question is this one: do the nerves which set into action the limbs of the animal experimented

[1] Nansen. *Nordiskt. Medicinskt. Archiv.*, 1887, xix. 4, p. 1-24.

upon possess a centre of whichever nature 'sensori-motor,' or 'kinæsthetic,' or purely 'motor,' &c.—always well defined, occupying in every brain of the same species of animal, the same area of the Rolandic convolutions? To this question I can give a more qualified answer, and will do so by means of a few chosen, and I consider, crucial experiments as well as by means of some facts gathered from human pathology.

Having sliced off the 'motor' area in the brain of a rabbit[1] I allowed it to rest for some time, and then performing Kussmaul and Turner's experiment of ligaturing carotid and vertebral vessels, I find that epileptic convulsions supervene at the same time in all four limbs.

In another experiment, also on a rabbit, only one motor area is destroyed, and it is found that the results of Kussmaul and Tenner's experiment are also identical in all four limbs.

In a dog[2] I performed the operation of removal of the sigmoid gyri on both hemispheres in Feb., 1886, and in April of the same year it was examined by a committee of the Biological Society and the report drawn up by Prof. Mathias Duval. I extract: "The gait appears very slightly choreic, that is to say that the animal walks balancing on either side, rather undulating like the tail of a fish; the hind limbs are slightly apart—in fact, very little disturbance, since the anterior limbs are perfectly normal. The left eye, which was the more troubled, permits good vision enough to avoid some obstacles.[3] Placed upon a table the animal jumps on the floor after the manner of a dog in the natural condition. As the examination fatigues him (by their duration) and worries him, he walks a little less well, slipping slightly upon the plank floor, the limbs apart slightly. Hearing is good. A lump of sugar being thrown at him he picks it up and searches for scattered particles on the floor. Examination with Dubois-Reymond's coil gives, when the coil is 10c. removed from

[1] Dupuy. "Physiology of the Brain," &c. *New York Medical Journal*, May, 1877.

[2] Mathias Duval, Rapport, &c. *C. R. Soc. de Biol.*, p. 371, 1886.

[3] There are always visual troubles in experiments on the cortex.

the core, good sensation in the right limb; at 30c. the same limb feels still but slightly; on the left limb he feels even a little with a removal of the coil to 40c. The hind limb (left) feels very well with coil at 30c., the right is still more sensitive with the coil at 30c. Electric irritation is therefore better felt on the left fore limb and right hind limb, as compared with the right fore and left hind limbs. By pressure with forceps sensation appears to be a little less on the right than on the left limb. The fore limbs feel equally well. That dog appears to be rather sensitive, or simply perhaps a little 'douillet.' It is killed by section of the abdominal aorta. . . . The brain, on examination, is shown to be adherent to the skin healed over the original wound, and by means of cicatricial tissue. On the right hemisphere there is a scar covering the three-fourths external posterior of the sigmoid gyrus; that scar covers also the convolutions immediately in front and behind. On the left hemisphere the scar is alike, only a little more extended in the antero-posterior area. . . . At the level of the scars the cerebral matter is rather much depressed, the border drawn towards the centre. After maceration in alcohol sections are made, which show on the right hemisphere ('il saute aux yeux') that the sigmoid gyrus has been completely removed, in the place of its deep portion (sulcus) cicatricial tissue is discovered which invades into the white central matter. On the left hemisphere the sigmoid gyrus grey substance appears also to have been completely removed, but the subjacent cicatricial tissue extends much less in depth, and part of the white matter of the gyrus appears to have been spared. Microscopic examination shows the fibroid cicatricial tissue infiltrated by granular elements. As said above, it is seen also that the scar on the left gyrus was more extended antero-posteriorly and, in fact, the sections show that the grey substance has been removed from the frontal convolution immediately in front of the gyrus."

Another dog was subsequently operated upon by me and shown to the Biological Society at several later meetings, and the results were in every manner similar. The brain was examined by Mr. Vignal. I have not alluded chiefly to the

experiments and results of Goltz because the reports of these published by the English Committee, as well as other reports published at Berlin later, show that although he has removed large areas of cortex, he has nevertheless, spared portions of the sigmoid gyrus cortex in one or in both hemispheres. But the brain examined by the Biological Society Committee, as said above, showed that the two gyri have been completely removed as I announced. In human pathology, since the new era of operating upon 'motor centres' has begun in earnest, literature records very interesting observations—I find a number of illustrations of facts identical with those obtained by my experiment on the dog. The following are taken of a greater number collected from journals, periodicals and memoirs easily obtainable:—

(1) Epilepsy induced by focal lesion in facio-lingual "motor centre." Operation performed. Removal of cyst from lower part of ascending frontal, size of a filbert, and situated partly in cortical and partly in white substances of the brain, and surrounded by a narrow zone of encephalitis. The muscles were paralysed after the fits (tongue, right facial, and platysma muscle). Complete recovery after operation.[1]

(2) Left-sided paralysis, arm and leg. Operation. Removal of gummous fluid with pultaceous particles and osteoplates. Later, second operation. Power of arm and leg restored so as to walk, perform household duties.[2]

(3) Paralysis of left hand and wrist, and epileptic. Operation upon cortex at level of fissure of Rolando but posteriorly, a piece of bone is removed which compressed the brain, which was found altered. A cyst is removed, and also about two teaspoonfuls of cerebral substance. After five days the patient was in good state. Paralysis relieved.[3]

(4) Convulsions, later hemiplegia, right side. Half an ounce of pus is removed from the ascending frontal incised one inch deep. Motor power recovered in arm and leg.[4]

[1] McEwen. *Lancet*, p. 257-8, 1888, with figure.
[2] *Id.*
[3] Keen. *Internat. Journal of Med. Science*, Nov. 4, 1888.
[4] *Liverpool Medico-Chirurgical Journal*, p. 303 1888.

(5) Epilepsy and left hemiplegia. Middle portion of fissure of Rolando is laid bare, and meninges and cortex diseased are removed. Complete recovery. Recovery of motor power.[1]

(6) Paresis of right leg, contracture of right hand, and epilepsy. Operation. Excision of lining of old lesion occupying substance of the ascending frontal. The excision was wide. Relieved from contracture and recovery, in a great measure of paresis.[2]

(7) Epilepsy. Right arm enfeebled after attack. Operation. 'Motor centre' circular incision made five millimetres around a cavity found in cerebral substance, and which is five centimetres deep and one centimetre wide. Paralysis disappeared three months later.[3]

(8) Epilepsy, right hemiplegia. Operation on superior third of ascending frontal. Very vascular scar three by two centimetres. It is excised together with five millimetres of surrounding substance and a little over two centimetres in depth. Relief from paralysis.[4]

Although I am dealing only with "motor centre doctrines" as "illustrated" by experiments made upon animals and surgical operations upon men, I will give some cases of disease of the "motor region" in man, since it is claimed by the anatomo-pathological school that nature performs far more precious experiments than experimenters can in their laboratories. The following are quoted from a number:—

(1) Atrophy of the upper thirds of left ascending convolutions. No paralysis right or left limbs.[5]

(2) Sarcoma growing from dura having apparently destroyed the greater part of motor area on right side, so far as one could judge with naked eye, the whole of motor centres for face and upper limb are destroyed. Microscopical examination shows that grey substance of this region seems

[1] *Congres français Chirurgie*, p. 308, 1885-6.
[2] *Bulletin de l'Academie de Medecin*, 1889, Aout. Lucas Championnière.
[3] Horsley. *British Med. Journal*, p. 674, 1886.
[4] *Id.* p. 672.
[5] Magnan. *C. R. Societ. de Biologie: Memoirs*, vol. xxx., p. 73, 1878.

to have completely disappeared, and yet there was absolutely no paralysis.[1]

As a number of cases, great indeed, can be found in literature, I will not quote more here, and these I have taken from Anglo-American literature mostly, as I am addressing an English Association.

Although aphasia is not my subject, yet since Broca's convolution is considered to be the seat of motor aphasia *par excellence*, and in the absence of which no speech can take place, and whereas it is taught that word-blindness, word-deafness, may exist with conservation of speech, if the occipital or temporo-sphenoidal lobes alone are altered, yet speech expression only persists so long as Broca's region is not destroyed.

(1) Patient was stricken down with right hemiplegia and aphasia eight years previously. She began to speak again at once, and in some manner suddenly, without having undergone preparatory re-education. About eighteen months after the attack of hemiplegia she could speak in a very precise manner—intellect intact. Autopsy. The left lobe is softened partly in the vascularisation area of the sylvian artery. The posterior portion of the third frontal (left) has been destroyed by softening, as also the insula, the junction of ascending frontal and parietal convolutions, and the first parietal are gone. In a word, the whole of the cortex of the lobe which make up the insula and marginal convolutions around it is destroyed. Other parts of the lobe are normal except grey substance of the corpus striatum, extra ventricular nucleus, which was destroyed. Intra-ventricular nucleus sound, marked atrophy of thalamus.[2]

(2) Paralysis of right upper and lower limbs, articulation thick and indistinct, patient complained of pain in paralysed limbs, emotional, noisy at night, &c. Left hemisphere sectioned, corpus striatum found softened to diffluence, also contiguous part of thalamus in limited area; softening involving from corpus striatum outward and upwards to surface including posterior part of third left frontal convolution.[3]

[1] Byrom Bramwell. *Brit. Med. Journ.*, April 21, 1888. Plate.
[2] Luys, in *C. R. Soc. de Biologie*, p. 330, vol. 28, 1876.
[3] Allan. *Lancet*, May 2nd, 1885, p. 797.

(3) Patient (cardiac dropsy) not the least symptom of any defect of speech, nothing pointing to cerebral mischief, when spoken to would give correct and sensible replies. Autopsy. Atheromatous arteries. Third left frontal convolution almost entirely destroyed, along with two-thirds of island of Reil. Over this area pia mater disorganised and converted into non-vascular transparent shreddy membrane, adherent to pulpy-brown surface of irregular excavation where brain substance was missing, brain otherwise apparently free from disease.[1]

(4) Patient suffering for three years from spasms of right side of face and later right upper limb, also lower limb; epileptic attacks. Answered questions rationally, though speech was thick. Autopsy. Medullary substance of left hemisphere reddened, line of cortex very indistinct, brain almost diffluent. A portion of this hemisphere (left), three inches long and one and a quarter deep, was thus affected. The ascending frontal and ascending parietal convolutions were implicated, the disease extending inward to the lateral ventricle; microscopic examination showed that glioma was the disease.[2]

There are a number of other cases of destruction of Broca's centre without aphasia (in right-handed persons) and even in Nothnagel's treatise on Cerebral Localisation (French translation) very fine examples are to be found, but he explains them by saying that aphasia did exist and was recovered from. That would according to him, confirm the rule.[3] It all depends upon the manner of viewing the case, as I shall show later on.

It is, unfortunately, impossible to study aphasia by means of experiments upon animals, but it is fortunately possible to do so by means of surgical operations performed

[1] Dr. Foulis. *Brit. Med. Journ.*, March 15th, 1879, p. 383.

[2] Cheesman. "Archives of Medicine," p. 84, t. 2, vol. 6. Plate. I quote this case, although the author makes no mention of aphasia lesion of the third frontal, because the context shows that destruction of Broca's centre existed, as also demonstrated by the delineation of the lesion in the plate. It is an "impartial" case published for a purpose not connected with aphasia.

[3] Nothnagel. "Traité clinique du diagnostic des Maladies des encéphale," p. 393.

upon man if I may trust to the accuracy of the following report :—

Three patients, under the charge of Burckardt were suffering from brain disease with that form of delirium present, which ends ordinarily in dementia. B. trephined with the idea of stopping the hallucinations by suppressing the speech centre in Broca's convolution. The desired result was obtained and sensory aphasia, consequent upon the operation, disappeared rapidly in all cases. One patient became more quiet and able to work without being dement. A second patient was also improved, the third remained partially aphasic, and after leaving the asylum committed suicide.[1] I am aware, of course, that those cases of (motor) aphasia I have quoted are considered to prove not only that Broca's speech centre is the "one," but also that on it becoming impaired, the other, in the opposite hemisphere, comes into activity. Some experiments I will report further on do not afford much foundation for that theory.

Another question I must speak of is the one concerning the absolute necessity of the cortex to produce true epileptic convulsions—it is a corollary of the question of "motor centre" naturally.

I have already alluded to some experiments which I performed long ago, to show that the limbs of an animal can become convulsed at the same time and in the same manner as when the cortex is present even after removal, partial or total, of the 'cortex motor' area.

(1) Having laid bare the brain of a dog, I proceed to induce epilepsy by means of electrical irritation of one 'motor area,' and later on I slice off the cortex from 'motor area' of both hemispheres. Electric irritation produces, when applied upon the surface of white substance thus laid bare, epileptic convulsions identical with those observed in the first experiment.

After allowing the animal some time to rest, a further slicing off of the brain (both sides) is made, so that nothing remains in the cranial cavity but the large ganglia attached

[1] *Revue des Sciences Medicales* (Hayem's) t. 37, p. 592.

still to the pons. This last organ being irritated with electricity, epileptic convulsions supervene; very violent, and, in one instance, return without electrical irritation.

(2) In several animals (dogs), having sliced off the motor areas in both hemispheres, essence of absinthe is injected (into saphena vein); a most violent epileptic fit breaks out and the animal continues for four minutes to have fits with short intermissions. The brain is sliced off completely and the pons irritated as before, and epileptic fits again occur.

These results were to be expected from previous experience. It is many years now since Brown-Séquard has shown that even after removing the brain of guinea-pigs, which he had rendered epileptic by lesions of the cord or of the sciatic nerve, fits did and would occur. I have made the same experiments a number of times already long ago, and it is therefore to be expected by me *a fortiori*, that true epileptic attacks will come on in animals, even in the absence of the cortex of motor centre area, since in some experiments the brain proper having been cut off attacks broke out.

Human pathology offers similar observations, since there are many records of epileptic fits beginning in the limbs, the 'motor centres' of which having been found destroyed by disease. Seppili[1] observed a case of which the record is: "Patient 30 years old, epileptic for seventeen years; left hemiatrophy of arm which is completely paralysed; leg moves slightly, both contracted. The attacks begin by a feeling of formication in the fingers of left hand, at the same time almost clonic convulsion of the arm muscles occur, then the left leg is involved, and face (left side) without loss of consciousness; later, the other side of the body becomes involved. Sometimes the fit begins at the same time in the two sides of the body, and then there is loss of consciousness. After the fit the left limbs remain completely flaccid. Autopsy. Left hemisphere sound, right hemisphere smaller, membranes adherent over large area of the right hemisphere comprising the posterior portion of the three frontal and

[1] In Soury. "Fonctions du Cerveau," p. 421.

the two ascending convolutions, the paracentral lobule and the parietal convolutions. The lesions extended into white substance and reached the superior and external parietes of the right lateral ventricle. Consistency of brain substance very feeble in this zone. The cortex as well as medullary tracts were transformed into fibrous tissue formed by numerous partitions which, by intersecting, formed small cavities containing semi-gelatinous substance. Microscopic examination showed that no nervous elements existed in that zone, but a considerable amount of amyloid corpuscles and pigmented and fatty granulations. The membrane which gave to this region the aspect of areolar tissue was formed by connective elements; numerous sclerosed vessels existed in it, but the lumen obstructed by accumulated pigments. Moreover, there was degenescence of right pyramidal tract, traced into right internal capsule, cerebral peduncle, bulbar pyramid and left lateral column of cord; opto-striated bodies normal."

Seppili being a distinguished physician and one of those who hold the 'Doctrine of Localisation' to be so well founded, I consider this case very instructive.

Is it possible to explain away the cases of cortex lesion in the 'motor area,' say for one limb without paralysis existing on the hypothesis of vicarious function of the neighbourhood or of the cortical area of the same 'centre' in the other hemisphere? Although Brown-Séquard has shown by experiment that the divided fibres of corpus callosum are excitable, giving rise to motor action in one or the other hemisphere according to the surface of the section irritated; still the balance of evidence is against the hypothesis that 'suppleance' or vicarious action takes place in the 'motor centre' of the other hemisphere. Carville and Duret[1] have found that, if some time after destruction of 'motor centres' for the limbs of one side of an animal has been made and the animal has recovered from the awkwardness or paresis, the 'motor centres' for the same limbs of the other side of the body, and situated in the other hemisphere, be also destroyed, instead of a paralysis following in

[1] Carville and Duret. *Archives de Physiologie*, 1875, p.

the limbs at first the seat of motor alteration and which had recovered, the new operation induces motor trouble in the limbs which were not affected by the first lesion. I have showed already that destruction of "motor centres" in both hemispheres is not followed by paralysis in either side of the body. And moreover, if after the animal having lost "motor centres" say the sigmoid gyrus in one side and having recovered and using all four limbs equally well, the brain is again laid bare and electrical irritation applied to the cortex all around the scar and in healthy cortex therefore, no motor effect is observed to follow in that limb—I have shown[1] also that if electricity be applied to the white substance surface produced by the destruction of the cortex several days after the healing process is complete, no motor effect is obtained and yet in those instances of absence of cortex no paralysis is observed.

It is evident from these that the cortex has no vicarious activity, that the "centres" do not replace one another in the same hemisphere, or in the other hemisphere—a conclusion at which I am not at all astonished, as I do not consider that the cortex contains "centres" which govern motion at all in the sense admitted by most. It does not seem possible to admit that motor impulses are generated in the so-called "motor cells" of the cortex and travel necessarily through and into the pyramidal tracts and cross into the cord. The reasons are the following, as found by experiment:

I divide one cerebral peduncle in a dog, and after allowing the animal to rest, the "motor area" situated in the hemisphere of the same side is irritated with electricity. A complete epileptic fit occurs, beginning in the side of the body opposite to peduncle divided.[2] Brown Séquard[3] had already found that after section of one cerebral peduncle or one half of the pons, electrical irritation of the "motor centre" of the same side was followed by motor action on

[1] Dupuy. *C. R. Soc. de Biol.*, 1875, in *Gazette Medicale de Paris*, 1875, p. 376.

[2] Dupuy. *C. R. Soc. de Biol.* for 1886, p. 19.

[3] Brown-Séquard. Specially reported in *Archives de Physiologie*, 1889, p. 220.

the opposite side of the body. It is also demonstrated experimentally that after hemisection of the cord and recovery from the effects of trauma and paralysis, animals recover motor power in the parts which had been incapacitated, and I have found that if hemisection on the two halves of the cord (alternate sections) be made below the bulb at some little distance from each other, then electrical excitation of left or right cortical "motor area" or sigmoid gyrus of the dog, motor action takes place in the limbs below the sections.

It remains now to report another series of experiments. I have found that it is possible to induce epileptic convulsions in a dog even after electrical irritation of the brain fails to do so; as for instance when in the etherized dog (after it has been subjected to cortical irritation by electricity), morphine is injected so as to induce more complete anæsthesia, then the current applied to the cortical "motor area" fails to induce any motor action; the same irritation however applied to the dura is followed by an epileptic attack.[1]

From the facts and arguments I have so far considered it does appear to me that the cortex of the "motor centres" are not necessarily the "centres" from which motor impulses are sent to the limbs, and before closing this line of argument I will report one last experiment.

Having laid bare the brain of a monkey under ether and morphine, the dura is split over one lobe so as to expose the "motor centre" for the opposite fore limb, and electrical irritation of that "centre" induces a fit of genuine Jacksonian epilepsy, and again, a second application induces a second attack, both having started and being localised in the same side, *i.e.*, opposite to the 'centre' irritated, but during the second attack the animal has recovered enough from the effects of narcotisation, and with the limbs not affected he grasps the sleeves of my coat so that by one half of his body he is holding on while the other is still epileptic. At this moment the fold of the dura which had been split and turned

[1] Dupuy. *C. R. Soc. de Biologie*, p. 339, vol. 41, 1889.

over to expose the 'motor centre' is irritated with the same electric current, and another fit of Jacksonian epilepsy follows, but this time in the limbs, with which the monkey was holding on. He looses his grasp, and while this second fit is going on the first is already recovered from.[1] Similar experiments made on a dog and on a cat gave the same results, and also the fits in some instances have become general after irritation of the dura, the cortex being inactive, a not unfrequent occurrence in Jacksonian epilepsy, the case quoted above from Seppili being an illustration in point, the patient had fits which were sometimes unilateral and sometimes general. It cannot be held, it appears to me, that the cells of the cortex of the brain constitute "motor centres." The experiments I have reported and the pathological facts do not substantiate that doctrine, nor are these elements "sensation centres." Since Tripier's experiments, a great number of cases are to be found in literature, showing alterations of sensation associated with lesions of the 'motor centres,' but a great number of cases are also to be found in which no such associations are to be found. In many of my experiments I have observed alterations of sensation after lesions of the 'motor centres'; also alterations of the sense of sight, and more commonly in the dog I may say that almost always the sense of sight is affected when lesions are made even on the frontal lobes. But many times also no such alteration of sensation is observed, and a "centre" to be specific must always appear at least endowed with the same faculty surely?

That the cells of the cortex do not cluster into well-defined 'centres' of innervation in the sense attached to the word 'centre' by those who teach the 'Doctrine of Localisation' is to me certain, and if all grant that the cortex contains 'centres' at all, then Brown-Séquard's well-known doctrine that centres are diffused through the whole cortical area, with possibly some 'spots' ordinarily more active;[2] a doctrine which he has been maintaining

[1] Dupuy. *C. R. Soc. de Biol.*, p. 274, t. 39, 1889.

[2] Little is known about the functions of the brain, but we can perceive that as the intellectual powers become highly developed, the various parts of

for a long time, and which is certainly supported by an immense number of observations ought to be the one accepted by those who believe the Localisation doctrine. Ferrier[1] has very clearly stated the case; "Causation is not established unless an invariable and unconditional relationship has been proved to exist between a particular lesion and a particular symptom." But if equally clearly expressed, the following statement: "In the case of the motor area it has been satisfactorily demonstrated that a destructive lesion invariably gives rise to motor paralysis, according to the position and extent of the lesion," is not proven by the experimental results and pathological observations, which I have laid before the "Association" to-night.

Before ending I beg leave to say a few words on an important question. It is apparent from the line of argument I have followed that I do not admit that the pyramidal tracts are the necessary connecting links between the cortex and the outgoing nerves of the cord. It is necessary to give some explanations. The experiments quoted of double (alternate) hemisection of the cord, but at different levels already goes some way to substantiate my theory; but there are so many cases of descending degeneration occupying the pyramidal tract alone that the demonstration of the crossed action of the motor tracts seems unshaken. Vulpian[2] has stated very clearly that this argument is not very strong. He gives instances in which he had seen atrophy of the white fibres of the anterior pyramids in a part of that tract, and yet above and below that atrophied portion the pyramidal fibres had a normal appearance. He gives two cases of deep lesions of the pyramids without paralysis. His statement "that voluntary incitations are not transmitted necessarily and conclusively to each side of the body by the anterior

the brain must be connected by very intricate channels of the freest communication, and as a consequence each separate part would perhaps tend to be less well fitted to answer to particular sensations or associations in a definite and inherited—that is instinctive—manner.—Darwin, *Descent of Man*, p. 68, second edition, 1888.

[1] Ferrier. "Functions of the Brain," second edition, p. 378.

[2] Vulpian. "Physiologie Generale and Comparée du Systeme Nerveux," p. 474 and 494.

pyramid of the opposite side" is fully borne out. A great number of experiments made by Vulpian himself, Schiff and Brown-Séquard, and myself, consisting in sections of the pyramids, and in which it was found that they are not, as Vulpian says, "exclusively and necessarily" the channels of transmission of voluntary incitations to the opposite of the body. There are a certain number of cases in literature, in which sclerosis of the bulb had destroyed one pyramid without there being a consequent paralysis.

From all I have said and all I know, I am able to state that in grey matter of the cerebro-spinal system there is no specified localisation of functions, in the sense understood by the majority of physiologists and all physicians. I believe that the grey tube element is *par excellence* the "matter" into which all the activities of the body centre; but I hold that it is inexcitable except through nerve fibres, and that it is neither 'motor' nor 'sensory'—it is potential substance. These functions of motion and sensation are the attributes of peripheric elements alone. Central grey substance simply reacts according to the nature of the impulse. Paralysis of motor activity [and of sensation] only takes place *directly* when there is destruction of the strands of fibres about to leave the cord, or leaving the cord and going to the muscles. Even when the so-called "motor cells" are destroyed or the seat of a lesion, paralysis which may appear is owing to another cause than loss of a "motor centre."[1]

[1] Vulpian. A woman, about sixty years old, and in whose left leg and foot the muscles were the seat of complete fatty atrophy—probably since childhood. Nervous fibres of muscular nerves less numerous than usual, and of smaller diameter generally, showed, nevertheless, all the characteristics of normal state, and such also was the case with those of the anterior roots to which they belonged. In this case there was well pronounced asymetry of dorso-lumbar enlargement of the cord, the left half was manifestly smaller than the right. Upon section made at that level after hardening in chromic acid, Mr. Prevost, my 'interne,' found and made me see that the cells of the anterior horn of the left side had disappeared, chiefly in the external portion of that horn, where not a single one was visible ("Physiologie du Systeme Nerveux," p. 345).

If any anatomist could see as I have seen the No. viii. fibres absolutely normal in the area of Deiter's nucleus while almost all the cells were absent, he would begin to reflect on the uncertainty of the simple study of normal sections, however carefully or conscientiously carried out (Seguin, "Archives of Medicine," t. 2, vol. x., p. 261). He is speaking of Von Guddens's preparations.

The grey substance central organ of the nervous system is everywhere identical in all its portions; impressions therein arriving are differentiated by the nature of their cause—that is, they arrive under differentiated specie from peripheric elements and the diffusion of impression is more or less great, is local or general, according as the measure of the exciting activity is weak or strong; but the 'nerve cells' have nothing to do with this.[1] When the grey substance is sectioned transversely it is obvious that the part below the section will only react under stimulus from the organs with which it has connection; and it can be segmented into several 'central organs' each independent of the other, but so long as there exists even a very small connecting link between segments of the grey substance, diffusion of impulses can take place all through, and objective phenomena become manifest as well as subjective. Now to speak of 'localisation of functions' in the cerebral cortex, and 'localisation of cerebral diseases,' appears to me only a convenient mode of expression, chiefly when used by clinicians, but devoid of any sense when used to express a physiological fact. According to Ferrier's[2] statement respecting the supposed sensory function ascribed by some to 'motor centres': "for a single case of destructive lesion of the motor area without anæsthesia is sufficient to overthrow the apparent casual connection founded on a host of positive instances," it is natural and logical that the same should hold good for the motor functions of the so-called 'motor centre' in the Rolandic area of the cortex also. If I am not mistaken, that one "single instance" means a plurality, to say the least!

My subject does not include the necessity of explaining in full how brain lesions produce objective motor symptoms, but a few words are not amiss, and with these I shall have ended. Since a motor paralysis manifest at one time dis-

[1] I need not here recall the works of Golgi and his school, and chiefly of Nausen. According to these, the cylinders-axis nerve fibres are directly connected by lateral prolongations, and reflex action takes place without passing through the cells. I know how hazardous it is to construe physiological theories upon anatomical researches however!

[2] Ferrier. "Functions of the Brain," p. 378, second edition.

appears, and the cortex material lesion with which it has been connected empirically and clinically still persists, it becomes logically evident that the presence of the "centre" (the seat of the lesion) is not a necessity for the restoration of functional activity of the parts which were paralysed. Secondly, since the lesion situated in different area of the cortex is found, in a case of motor paralysis, involving the same parts of the body as in the previous observation, it is logically evident that empirically and clinically, diversely localised lesions may cause the same symptoms;[1] and if a lesion situated in a 'centre'—not the one which is said to be the "motor centre" for a given group of muscles—on being destroyed the group of muscles is found paralysed, that state of paralysis may be brought about by an influence which manifests the activities of nervous elements 'reflection,' the result of electric variation. All paralysis is the result of a reflex starting from the seat of the lesion wherever situated; in clinics, paralysis and convulsion involving a limb, and motor aphasia, are known to arise from irritations (organic, toxic, &c.) situated in the abdominal cavity sometimes. The affirmation, that in the case of hemiplegia or convulsion, epilepsy, motor aphasia, when owing to the action of a toxic agent in the system, or to the presence of worms in the bowels, do not last, while the same derangements of the motor apparatus are persistent in case of brain or 'motor centre lesion,' is so often repeated because unchallenged, besides it is irrelevent. I have given only a very few cases for the sake of brevity, but literature is rich in instances of motor derangements having disappeared, the lesion in the cortex 'motor centre' persisting. It is my intention to treat of the mechanism of paralysis from brain diseases at some future occasion; this evening I have occupied enough of the valuable time of the members of the Neurological Association.

[1] Brown-Séquard in *Lancet*, 1876-77.

ASCENDING DEGENERATIONS RESULTING FROM LESIONS OF THE SPINAL CORD IN MONKEYS.

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EXPERIMENTAL physiology has shown that the periphery of the lateral and anterior columns of the spinal cord in animals, such as rabbits, dogs, cats and monkeys, is made up of fibres which connect cells of the cord with the cerebellum, especially the vermis. The cells of Clarke's column have long been considered to be connected in this way by means of the direct cerebellar tract, and it was formerly considered by Flechsig and others that the column known as the antero-lateral tract of Gowers was a part of the ascending cerebellar tract. Bastian[1] described this connection in a case of paraplegia, and he considered the ascending antero-lateral degeneration to be a part of the direct cerebellar tract. From pathological observations (notably from a remarkable case of gun-shot injury), Gowers believes the antero-lateral column which bears his name to be connected with the transmission of painful sensations from the opposite side of the body. He states[2] "It has often been confounded with the cerebellar tract, and it is this which has given rise to the impression that the cerebellar tract extends further forwards than it really does. In the lumbar region, this tract lies across the lateral column, on a level with the posterior commissure from which its fibres probably come. This tract has recently been found by Bechterew to undergo development at a different period from the rest of the lateral

[1] *Med. Chr. Trans.*, 1867.

[2] "Diseases of the Nervous System," vol. i., Spinal Cord and Nerves.

column. It is almost certainly a sensory tract, and physiological facts seem to show that its fibres are connected with the posterior roots of the opposite side. Fibres probably pass to it through the whole length of the cord, and these are mingled together, so that the degeneration arises from a lesion of the lower part of the cord."

Edinger[1] is also of opinion that the antero-lateral tract is connected with the transmission of painful sensations from the opposite side—maintaining however, that the fibres composing the tract are derived from cells of the posterior horn and that these fibres decussate in the anterior commissure. He supports the latter statement by microscopical investigations on the spinal cords of fishes, reptiles, and amphibians, also by reference to the experimental work of Auerbach, an abstract of whose work will shortly be given in this paper. It is right to state that Auerbach himself energetically repudiates conclusions drawn from his own work by Edinger.

Since section of the posterior roots in animals produces no degeneration in the antero-lateral column, it is highly improbable that the fibres of this tract are derived from any other sources than from cells in the spinal cord, notwithstanding the recent publication of Berdez,[2] of which a short abstract is given below. Experiment does not seem to show

[1] Einiges von Verlauf der Gefühlsbahnen im Centralen Nerven System. Dr. L. Edinger, *Deutsche Medicinishe Wochenschrift*, 1890.

[2] Recherches experimentales sur le trajet des fibres centripètes dans la Moëlle Epinière. Par le Dr. Berdez. *Revue Med. de la Suisse Romande*, 1892.

Berdez divided roots on one side in guinea-pigs. Some of the lesions injured also the posterior horn and posterior column. The sections were stained by Marchi's method. A scattered degeneration in both antero-lateral columns was observed above the lesion, nearly equal on the two sides, but the higher one goes the fewer the degenerated fibres seen. Bilateral degeneration of the posterior columns occurred but much more marked on the side of the lesion. The passage of fibres from one side to the other is made by the posterior part of the grey commissure. Berdez noted also the existence of degenerated descending fibres occupying the middle part of the posterior column. The existence also (but less numerous than the ascending) of descending degenerated fibres in the antero-lateral column.

In the cord of an animal so small as a guinea-pig it is very easy to produce damage, and scattered degeneration results. Moreover scattered black points are not always degenerated fibres.

The guinea-pig is not a very satisfactory animal as it is generally considered that its tracts are not so well defined as those of animals higher in the zoological scale (F. W. M.)

what cells give rise to these fibres. The fibres are large in size, they have a long course, and presumably, therefore, the cells are large. It seems that many of these fibres come from cells distributed in the lumbar region of the cord. Clarke's column has been shown experimentally by me to give origin to the fibres of the direct cerebellar tract.[1] As I shall show, this antero-lateral tract must be considered the *ventral* portion of the ascending cerebellar tract. The question thus arises—do the fibres forming it *also* spring from the cells of Clarke's column? These cells are not found in the lumbar enlargement *below* the third segment, but it is possible that the fibre may take a circuitous course downwards, and then loop upwards in the lateral column before reaching the periphery of the cord, as we find they do at their termination. Thus a lesion of the lumbar part of the cord even below the situation of Clarke's column might give rise to degeneration in the ventral cerebellar tract, or another explanation is offered by the experiments of Singer[2] (of which a short abstract is given below), viz., that fibres in the antero-lateral column spring from cells of the anterior cornua

[1] Bi-polar cells of the spinal cord.—MOTT, *Brain*, 1891.

[2] Ueber die Veränderungen am Rückenmark nach zeitweiler Verschliessung der Bauch Aorta von J. Singer. *Akad. der Wissenschaften zu Wien, Sitzungsberichte*, 3 *Abtheilung*, 95-96, 1887.

Singer pursuing the investigations of Ehrlich and Brieger of compression of the abdominal aorta found certain changes to occur in the lumbar enlargement of the spinal cord.

Extra peritoneal compression of the aorta was made in animals just about where the left renal artery is given off. One animal was kept alive 5 weeks, the others were killed at shorter intervals of time after the operation. When the compression was made on the right side of the spine no results were obtained. The compression was maintained for one hour, and was followed by permanent paralysis and analgesia of the hinder extremities.

The resulting changes were briefly as follows:—At the end of 24-36 hours. Microscopical examination of sections of the lumbar spinal cord after suitable hardening and staining showed that the anterior cornual cells had commenced to undergo degenerative changes. The cells showed a fine granular change, and here and there around the vessels of the grey matter, escape of red corpuscles was observed.

Four days after operation there existed a well-marked granular degeneration of the anterior cornual cells, with breaking off and even absence of the processes, besides there were divers signs of change in the nucleus. The medullated fibres of the anterior horn were markedly varicose, and there were irregular clumps of myelin, and swollen axis cylinders scattered about. The white substance also showed numerous swollen axis cylinders.

Eight days after the operation. The anterior horns were smaller, the ganglion cells having disappeared. There was destruction of the medullary

in the lumbar enlargement. Testut[1] states; "the fibres constituting the cerebellar tract (which he divides into a ventral and dorsal portion) thus represent directly long anastomoses thrown between the vermis superior of the cerebellum and the different levels of the vesicular column of Clarke." That both the ventral and dorsal ascending cerebellar fibres terminate in the vermis superior, the following experimental evidence conclusively proves. See fig. 1, a diagrammatic representation, modified from Testut, of the termination of the dorsal and ventral ascending cerebellar fibres in the vermis superior.

Von Monakow,[2] quoted by Löwenthal,[3] made an almost perfect hemisection of the spinal cord immediately below the decussation of the pyramids in a new-born rabbit, and proved that a small portion of the lateral column is continued directly up into the region of the pons. The ascending atrophy of this bundle, which is compact and of considerable size in the rabbit, is situated between the ventral extremity of the ascending root of the fifth and the lateral nucleus. He followed it further to the level of origin of the fifth pair, and considered that it was continued into the lateral part of the ribbon of Reil, and he terms it "Aberrirende Seitenstrangs Bundel."

Löwenthal's admirable description of his discovery of the course of these fibres, which I have unfortunately only recently become acquainted with, clearly shows the destination of this tract. He made a spinal lesion in the dog between the fifth and sixth pairs of cervical nerves, destroying the whole left lateral column, and he figures the course of the ascending ventral and dorsal cerebellar tracts to their termination in the vermis. The conclusions that he comes to from his experiments are as follows:

sheath of fibres in the anterior horn and certain regions of the white matter.

Three weeks after the operation. The posterior roots, spinal ganglia and posterior columns were quite normal. The anterior horns greatly diminished in size, and a disappearance of the anterior cornual cells with replacement of the nerve network by connective tissue. The anterior and posterior commissure both intact. In the anterior and lateral columns, especially the outer periphery (situation of antero-lateral tract of Gower's) are numerous degenerated fibres. Below the lumbar enlargement the spinal cord appears normal. The anterior roots, however, are partially degenerated.

[1] "Traité d'anatomie humaine," vol. ii., p. 352.

[2] *Archiv. fur psychiatrie*, 1883.

[3] Degeneration secondaire ascendante dans le bulbe rachidien dans le pont, et dans l'étage supérieure de l'Isthme. *Revue Medicale de la Suisse Romande*, 1885.

the cerebellar bundle extends around the periphery of the spinal bulb in its lower part, from the ventral extremity of the substantia gelatinosa, up to the level of the antero-lateral nucleus. Its ultimate path is by two different courses—one part of its fibres is displaced more and more dorsally, and terminates in

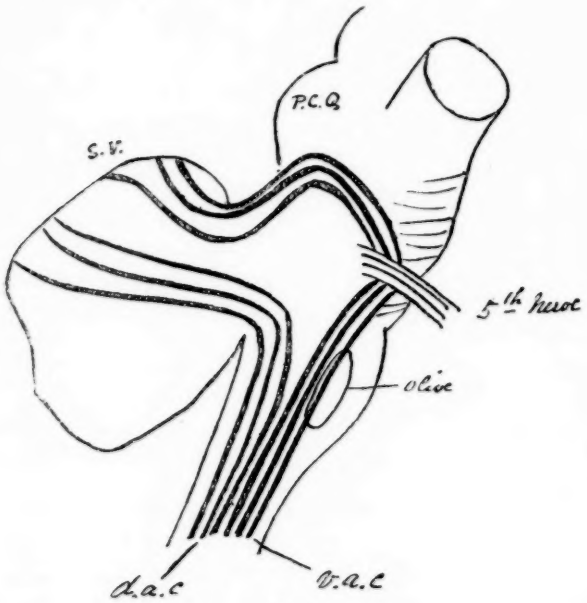


FIG. 1.

Diagrammatic representation of the course of the fibres of the ascending cerebellar tract. The ventral v.a.c. and dorsal portions d.a.c. are united in the cord and lower part of the medulla. The latter passes into the restiform body and terminates in the dorsal portion of the superior vermis s.v., the former becomes more ventral until it reaches the fifth nerve, when it forms a loop backwards to reach the posterior and outer surface of the superior peduncle. The fibres then course downwards and sweep round into the anterior portion of the superior vermis. PCQ, posterior corpora quadrigemina.

the restiform body (the dorsal portion of the direct cerebellar tract). He then gives reasons for connecting these fibres with the superior vermis.

Another portion of the cerebellar tract follows the ventro-lateral periphery of the medulla, running from behind, forwards, and always being directed more laterally (ventral portion of the

cerebellar tract). In the neighbourhood of the fifth pair of nerves it takes a deeper course, traversing the lateral region of the pons, and is covered by fibres of the middle cerebellar peduncle. A little distance behind the posterior Corpora Quadrigemina, it escapes on to the lateral furrow of the isthmus and turns towards the external periphery of the superior cerebellar peduncle, after which it runs backwards in a retrograde direction, describing outside of the said peduncle a prolonged half-turn, and comes to place itself successively on its external dorsal and dorso-internal side, which it occupies at the position where the superior cerebellar peduncle reaches the medullary velum of the cerebellum. The ultimate destination of this part of the cerebellar tract could not be elucidated. In cases where the lesion of the lateral column was limited to its dorsal half, the ascending degeneration of the cerebellar bundle could only be followed into the restiform body. The ventral portion remains intact. The fibres of the ventral portion of the cerebellar bundle only affect relations by contiguity during part of their course with the lower fillet.

Auerbach[1] destroyed the posterior part of one half the spinal cord for a considerable length, the lesion included the posterior white column, the posterior horn of grey matter and the posterior part of the lateral column. It was followed by degenerations above the lesion in the direct cerebellar tract and the antero-lateral of both sides, with degeneration of the anterior commissure. The number of the degenerated fibres of the anterior and antero-lateral columns decreases from below upwards; the fibres seem to pass into the grey matter, partly crossing at the anterior commissure. Even if the parts mentioned had been injured to a great extent in the lumbar portion of the cord (on one side only), the number of degenerated fibres is almost the same on both sides at the level of the decussation of the pyramids. The ventro-lateral cerebellar degeneration he traces up to the ventral part of the superior vermis—the dorso-lateral cerebellar tract he traces up to the dorsal portion of the superior vermis. He also makes out a third division, which originating in the lumbar cord, remains united with the ventral portion up to the level of exit of the fifth, when it enters the inner portion of the peduncle of the cerebellum (which peduncle I am unable to determine) and

[1] Zür Anatomie der aufsteigend Degeneriren den Systeme des Rückenmarks v. Dr. Auerbach. Anatomische Anzeige, 1890; also Zur Anatomie der Vorderseitenstrangreste. *Archiv. fur Pathologische Anatomie u. Physiologie* v. Virchow, v. 121, 1890.

courses towards the dentate nucleus. These fibres eventually pass from the corpus dentatum into the dorsal portion of the superior peduncle of the cerebellum. It is thus to be seen that Auerbach in a great measure confirms the observations of Löwenthal.

The previous hemisection experiments which I made upon monkeys[1] were not conducted with the view of tracing the degenerations, so much as to ascertain the physiological effects produced during life. The animals were kept a long time, and the resulting degeneration was a sclerosis. I was unable to trace the antero-lateral tract beyond the lateral nucleus in the lower part of the pons; neither was Tooth, who made hemisections in monkeys for the express purpose of tracing this degeneration, and he kept the animals alive only a short time.[2]

Prof. Schäfer having found the great advantage of Marchi's method in tracing degenerated fibres, made hemisections of the spinal cord in two monkeys with the express purpose of following out the resulting degenerations. He has kindly placed at my service the material, and the accompanying woodcut (fig. 2). One of his hemisections was made in the mid-dorsal region, and the other at the 11th to 12th dorsal segment. It is noteworthy that the animals, when tested as regards sensibility on the two sides, entirely corroborated (so *he* informs me) the results which I have published.[3] Microphoto i. shows the ascending degeneration resulting from the latter lesion.

My own experiments were undertaken with a view of tracing the antero-lateral tract to its destination, also of ascertaining the physiological effects of its division on one side and on both sides.

The antero-lateral tract was divided, either on one side or on both sides, by means of a delicate knife, the blade of which was at right angles to a steel shaft, the

[1] Results of hemisection of the Spinal Cord in Monkeys. *Phil. Trans. Royal Soc.*, v. 183.

[2] When I brought the following experimental evidence before the Physiological and Neurological Societies, Dr. Tooth stated that he had altered his opinion with regard to the destination of the antero-lateral tract, and said that his observations entirely corroborated those of Löwenthal in the dog and mine in the monkey.

[3] *Loc. cit.* 10.

cutting edge being in front. After exposing the spinal cord, the point of the knife was inserted into the middle of the lateral column, the edge of the knife was then pushed forward so as to divide the anterior half of one side of the cord. In one case, this was effected on the opposite side as well, so that an antero-hemisection was made, that is to say, the spinal cord was completely divided in front of the central canal. The wounds were dressed antiseptically, and in all cases healed readily by first intention.[1]

EXPERIMENT I.

A section of both antero-lateral columns was made in the mid-dorsal region. The left pyramidal tract was partially injured. The destruction of the anterior column was complete on the right side, and almost complete on the left. The next day after the operation the animal was tested; and, as far as I could ascertain by careful testing, with independent witnesses, no loss of painful sensation was appreciable. The animal responded as briskly to heat and pricking with a needle as before the operation; the only perceptible difference was a slight difficulty and delay in localising the presence of a clip placed on the foot or leg of the left side (that upon which the pyramidal degeneration existed). On the fourth day after the operation, the weakness of the left lower limb had passed off, and the animal behaved in all respects, as far as could be made out, like a normal monkey. It was killed at the end of three weeks with chloroform. There was most extensive degeneration on both sides in the antero-lateral columns above the lesion, also in the antero-lateral region below the lesion.

EXPERIMENT II.

Section of the right antero-lateral region of the spinal cord of the third cervical segment, and the lesion was very successful, as micro-photograph ii. shows. Half-an-

[1] The expenses of this research were partially defrayed by a Grant from the Brit. Med. Assoc.

hour after the injury, the animal had recovered consciousness, and could feel pricking and touching on both sides equally well; the pupils were equal, and both sides of the chest appeared to move normally. There was no vaso-motor paralysis, but the right arm and leg were partially paralysed. Subsequent microscopical examination showed that the cross pyramidal tract had been considerably injured.

After twenty-four hours only the *finer* movements of the hand and foot appeared much affected. Bi-lateral associated movements had already in a great measure returned on the right side. Sensation was tested as follows: the animal was prevented from seeing what I did, but when I fixed a weak clip upon the left foot (non-paralysed), it drew up its leg and removed it several times in succession. I then placed the clip upon a similar spot on the right foot, but the animal took no notice of its application. When fixed on the hand, it removed it with the teeth from both sides. When pricked with a needle, or touched with a test tube containing hot water, it gave evident signs of feeling on both sides. The animal, however, localised correctly the spot irritated on the left leg, but on the right it put down its hand, but nowhere near the place. Four days later these experiments were repeated, with the same results, and again, some few days later the animal was placed under æther, and a clip was fixed to the right foot (the side of the injury). When the animal came to, it made no attempt to remove it; whereas when fixed on the left foot, under æther, the clip was immediately removed after return of consciousness. It now feels pricking on both sides. The paralysis is much less marked, but there is some feebleness of grasp in the right hand and right foot. The animal was killed with chloroform, and the brain and spinal cord put into Müller's fluid, all precautions being taken to prevent decomposition occurring in the central parts. This animal seemed to show that localization of touch and pain is intimately connected with movement. I find that most psychologists support this view. Sully, p. 164, says in a foot-note:—"The fundamental idea here expounded that

the localization of touch-impressions and the tactual perception of space is acquired by the help of the experience of movement may be said to underly all recent attempts to trace the genesis of space perceptions. It is to be added that the German psychologists rightly emphasize the part played in the development of the perception of space by the extended surface of the skin, with its capability of yielding us at the same movement a number of locally distinct sensations."

Experiment III.

Section of the antero-lateral column on the left side, at the level of the fifth cervical segment, was made as in the previous case, care being taken to avoid, if possible, injury of the pyramidal tract. The next day the animal was tested and gave evident signs of feeling, both pricking and heat on both sides. There was slight paralysis of the arm and leg of the left side, but apparently no difficulty in localising the clip on either side. The animal alive three months after the operation, and still shows no symptoms of injury, except the scar on the skin. I have always made it a rule to test the animals before the operation, and this animal was not really a satisfactory one for the purpose of this experiment, for it did not always remove the clip nor even respond to pricking before the operation; so that no very definite inference can be drawn, although these three experiments tend to show that the inability to localise sensation is connected with injury to the posterior "*pyramidal*" portion of the lateral column. They certainly tend to negative the view that the antero-lateral tract conveys painful impulses coming from the opposite side of the body. Moreover, this view is not in accordance with the destination of this tract. Cases of compression of the cord occur in which not a single healthy fibre can be seen in the sections, and yet painful impressions, such as pricking, can be felt acutely but not localised; and cases of syringomyelia seem to show that the grey matter conducts painful sensations, as first determined experimentally by Schiff. Moreover, Singer's experiments tend to show that the grey matter conducts painful sensations.

Experiment IV.

A section of the left posterior column, the posterior horn and the posterior part of the lateral column was made between the first and second lumbar. This really corresponds to Auerbach's experiment, except that he destroyed it in a much greater length, therefore I do not attribute great importance to it. The effect of this lesion which extended for about a quarter inch was to produce weakness in the left foot, and loss of localisation to a great extent. I tested the animal in the usual manner. It felt painful sensations, but was unable to localise a clip placed on the foot, neither did it remove it upon regaining consciousness when the clip had been fixed on the foot under an anæsthetic. I am at present engaged upon further experiments of this nature. Examination of the cord shewed, however, above the lesion a scattered degeneration in both anterior and lateral columns, which gradually became less and less as the cervical region was approached. At the level of the second cervical the number of degenerated fibres in the ventral cerebellar tract of the same side were more than twice as numerous as on the opposite side to the lesion.

To revert to the degenerations resulting from Professor Schäfer's hemisections and my own experimental sections of the antero-lateral portion of the cord, it may be stated that the brain and spinal cord in these animals with the exception of Experiment III., were hardened in Müller's fluid, and the sections stained by Marchi's[1] method. The accompanying woodcut shews the degeneration above and below the lesion following a hemisection at the eleventh and twelfth dorsal. The black dots indicate the degenerated fibres. The lumbar section shews the degeneration below the lesion in the antero-lateral periphery of the cord. These descending fibres can be traced as I have shewn,[2] down to the extreme termination of the coccygeal portion of the cord.

[1] Small pieces of the cord, &c., are placed in a solution of 1 part 1 per cent. osmic acid, and 2 parts 3 per cent. bichromate solution for a week in a warm chamber, the temperature never exceeding 37° C. The pieces are then embedded in celloidin, and thin sections cut and mounted in canada balsam. This method is perfectly reliable, if certain precautions are taken in preparation and observation.

[2] *Loc. cit.*

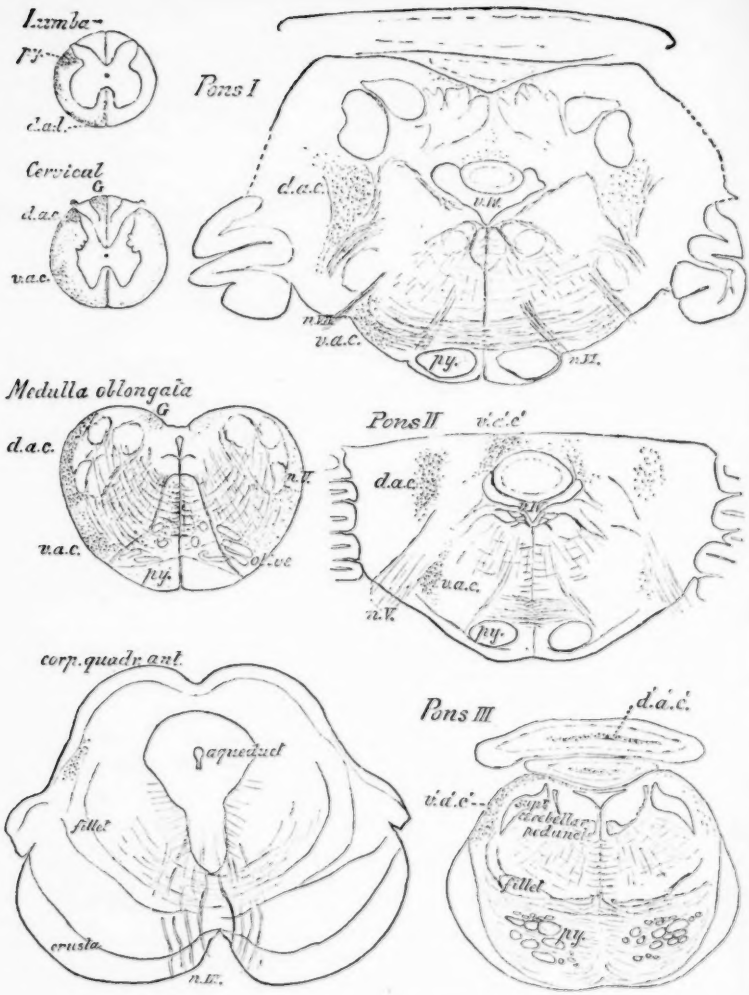


FIG. 2.—DEGENERATIONS IN THE SPINAL CORD, MEDULLA OBLONGATA, PONS VAROLII AND MESENCEPHALON OF A MONKEY FOLLOWING HEMISECTION AT THE 12TH DORSAL NERVE (E. A. S.).

The hemisection was on the left side of the cord and was complete. The section outlines drawn to a scale with a camera lucida. The degenerated fibres shown by black dots. Those in the section of the lumbar cord are descending, all the rest are ascending. *py.*, pyramidal tract; *d.a.l.*, descending antero-lateral tract; *d.a.c.*, dorso-lateral ascending cerebellar tract; *d'.a'.c'.* (in Pons. III.), degeneration of fibres of this tract in the white matter of the cerebellar worm; *v.a.c.*, ventro-lateral ascending cerebellar tract; *v'.a'.c'.* (in Pons II. and III.), degenerated fibres of this tract passing dorsally into the valve of Vieussens (in Pons III.) and into the white matter of the vermis (in Pons II.); *n.III.*, *n.V.*, *n.VI.*, *n.VII.*, issuing fibres of the 3rd, 5th, 6th, and 7th nerve-roots; *v.IV.*, 4 h ventricle.

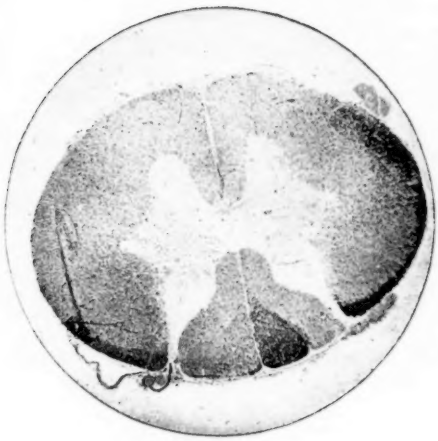
Some of them are undoubtedly long fibres, but Löwenthal[1] has shown that they do not, however, come from the cortex.

He terms this tract the *faisceau marginal*. My section of the anterior column at the third cervical segment (Experiment II.), produced a most extensive degeneration in this tract which could be traced right down to the termination of the cord. Some of the fibres seem to decussate in the anterior commissure, as the degeneration on the opposite side was more extensive low down than in the mid-dorsal region. They could be traced into the anterior cornua, possibly there they establish connections with the motor cells. Where *do* these fibres come from, if they do not come from the cortex, and therefore do not correspond to the direct pyramidal tract in man? Marchi[2] has answered this question by examining the cords of animals in which Luciani had produced lesions of the cerebellum, and asserts that efferent fibres from the cerebellum pass down the cord, mingled with the ascending antero-lateral and direct cerebellar tracts; so that we have the whole periphery of the anterior and lateral columns of the cord (for the most part) occupied by cerebellar fibres, some ascending and some descending. It is however possible and probable that many vertical fibres of a commissural nature to the cells of the cord at different levels exist even at the antero-lateral periphery mingled with the cerebellar fibres. With regard to ascending degeneration, nothing need be said concerning the course of the fibres of the column of Goll; this is well-known and generally accepted. Its course is shown in the figs. representing a section of the cervical region of the cord and the medulla. The ascending cerebellar tract is shown in the cervical region occupying the anterior and lateral periphery of the cord. The ventral and dorsal portions represented by v. a. c., d. a. c., are continuous, although each forms at one point a more compact and triangular area of degeneration. In micro-photograph iii., a section of the first cervical seg-

[1] *Revue Medicale de la Suisse Romande*, No. 9, 1886. La Région pyramidal de la capsule interne chez le chien et la constitution du cordon antero-laterale de la Moëlle par N. Löwenthal.

[2] *Ref. Neurolog. Centralblatt*, x. 8. Sullorigine e decorso dei peduncolari cerebellari e sui loro rapporti cogli altri centri nervosi pel Dr. Vittorio Marchi, Pubblicazione del R. Inst. di Studi Superiori in Firenze.

ment in Experiment II., the degeneration is almost entirely limited to the ventral portion. In the medulla, the two tracts are continuous, as shown in fig. 2, medulla oblongata; but in its upper part the two tend to become separated, as shown in pons I., the dorsal-cerebellar fibres become oblique, running backwards in the restiform body, while the ventral portion becomes more ventral and forms as shewn in micro-photographs iv. and v., islets of degeneration amidst the arciform fibres of the pons. These degenerated fibres, owing to their somewhat oblique direction outwards, appear somewhat larger than they do in the cord, and it may be noted that there are scattered degenerated fibres internal to the compact degenerated area. At the level of origin of the fifth nerve, as shown in fig. 2, pons II., the fibres become oblique, and form a loop over this nerve; and posteriorly where the isthmus of white substance connects the superior cerebellar peduncle with the cerebellum, degenerated fibres can be seen entering the anterior portion of the superior vermis (the roof nucleus of Stilling), fig. 2, v' a' c', and micro-photos vi. and vii. Still higher at pons III., just behind the posterior corpora quadrigemina, a tract of degenerated fibres is seen covering the superior peduncle of the cerebellum, and continuous with the valve of Vieussens, into which some few degenerated fibres run. Micro-photo vii. shows the triangular portion of these fibres under a high power. Still another figure shows a section of the anterior corpora quadrigemina, in which a few degenerated fibres of the fillet of the same side are shown; these I believe to be the scattered fibres which were observed internal to the compact area of degenerated ventral cerebellar fibres seen among the arciform fibres in the lower part of the pons. The dorsal cerebellar fibres could be traced from the restiform body into the dorsal portion of the superior vermis whilst the ventral portion was found as described by Löwenthal and Auerbach, to take a much more circuitous course. In the pons they leave their ventral situation, forming a loop over the fifth nerve, they are then directed obliquely upwards and backwards, on to the surface of the superior peduncle: forming a layer of fibres continuous with the valve



i.

Section of the spinal cord of 4th cervical segment. The degeneration is shown black in Goll's column, in the (direct) dorsal cerebellar and (antero-lateral) ventral cerebellar tracts in the right side. This degeneration resulted from a hemisection on the right side at 11th to 12th dorsal segment.

ii.

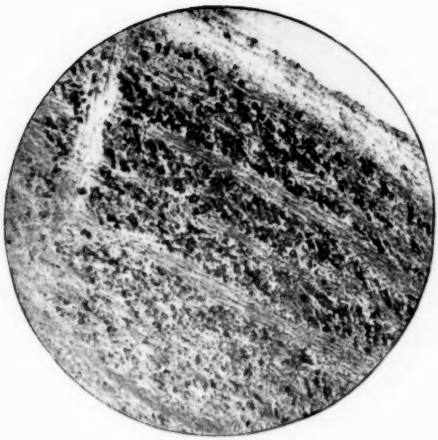
Destruction of the antero-lateral half of the cord on one side at the level of the 3rd cervical segment section near the lesion. Experiment II.

iii.

Section of the spinal cord at 1st cervical segment. The black on the left antero-lateral periphery is the ascending degeneration resulting from lesion shown in ii.

iv.

Degenerated ventral cerebellar fibres amidst the arciform fibres in the lower part of the pons, forming a compact tract between the 6th nerve and the ascending root of the 5th.



v.

The same as iv., more highly magnified. The fibres are just beginning to take an oblique direction.

vi.

The degenerated fibres, passing from the superior cerebellar peduncle into the ventral portion of the superior vermis.

vii.

The degenerated fibres, as they are passing through the isthmus into the vermis, more highly magnified than in the last vi.

viii.

The degenerated fibres of the ventral cerebellar tract shown, taking an oblique course backwards to reach the superior cerebellar peduncle. The portion shown in the photograph is $v' a' c'$. Pons III. Fig. 2.

of Vieussens and separated from the peduncle by a thin layer of gray matter, they then run downwards on the posterior surface of the peduncle as far as its junction with the cerebellum at the isthmus, where these degenerated fibres can be seen streaming inwards to the superior vermis. The peculiar tortuous course taken by these fibres is diagrammatically shown in fig. 1. On looking over my old preparations, prepared by Weigert's method, I can now follow in them the course of the degeneration of this tract,[1] although I previously stated that I was unable to trace the degeneration further than the lower border of the pons.

Summary.—The peripheral portion of the anterior and lateral columns consists in great part of ascending and descending cerebellar fibres. The former may be divided into a ventral and a dorsal portion, which may be termed the *ventral and dorsal ascending cerebellar tract*, instead of antero-lateral and direct cerebellar. The ventral portion may be completely divided in monkeys apparently without producing analgesia. The ascending cerebellar tract, consisting of ventral and dorsal portions forms a connecting bridge between the superior vermis of the cerebellum and cells in the cord. The dorsal portion connects the cells of Clarke's column with the dorsal portion of the superior vermis. The ventral portion connects certain cells of the cord with the ventral portion of the superior vermis. Section of the antero-lateral column in the cervical region produces a much denser degeneration amidst the arciform fibres than can be accounted for by the slight injury to the direct cerebellar fibres ; the *very extensive tract* of degeneration corresponding to Gower's antero-lateral tract, which, with Löwenthal, I agree should be called ventral cerebellar, has been traced in the monkey to the superior vermis by way of the superior cerebellar peduncle, forming in its course a remarkable loop over the 5th nerve, as shown in fig. 1. Scattered fibres belonging to the fillet can be traced as far as the anterior quadrigeminal body of the same side.

[1] The Micro-photos are, for the most part, taken from specimens made from the cord and brain of Case II., section of the antero-lateral column, at the third cervical segment. *Loc. cit.* Results of hemisection of the spinal cord in monkeys.

THE CHANGES IN THE OPTIC TRACTS AND CHIASMA, IN A CASE OF UNILATERAL OPTIC ATROPHY.

BY R. T. WILLIAMSON, M.D.(LOND.), M.R.C.P.

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IT is now allowed by all observers—with the exception of Michel—that a semi-decussation of the optic nerve fibres occurs at the chiasma, but as to the exact relation of the crossed and uncrossed fibres in the optic tracts and in the chiasma, considerable diversity of opinion exists.

On looking over the records of the microscopical examination of the optic tracts and chiasma, in cases in which unilateral blindness had existed for some time previous to death, one finds that the results vary. In many of the cases reported no definite *bundles* of degenerated fibres were found in the optic tracts. In two cases of unilateral optic atrophy, Gowers[1] found that there was a little excess of connective tissue in both optic tracts. The microscopical changes were nearly equally distributed through both tracts but the tract, on the side opposite the affected nerve was rather smaller than the other.

Baumgarten[2] and Burdach[3] report cases in which tracts of degeneration were traced for a short distance only beyond the chiasma.

In some of the cases recorded, bundles of degenerated fibres have been traced in the optic tracts.

Thus, Siemerling[4] found, in a case of unilateral blind-

[1] GOWERS, *Centralbl. f. die Med. Wissensch*, 1878, No. 31, S.562, and *Medical Ophthalmoscopy*, p. 118, third edition.
[2] BAUMGARTEN. *Centralbl. f. die Med. Wissensch*, 1878, No. 31.
[3] BURDACH v. *Graefe's Archiv. f. Ophth.*, xxix., 3 S. 135.
[4] SIEMERLING v. *Archiv f. Psychiatrie*, xix., S. 401.

ness, that the uncrossed bundle of fibres occupied a central position in the tract, but the boundary between crossed and uncrossed fibres was not sharply defined.

Purtscher[1] found a degenerated part in the centre of the tract on the same side as the blindness, and at the periphery of the opposite tract. v. Gudden[2] found in a case of total atrophy of one optic nerve, that the uncrossed fibres were more in the upper part of the tract, the crossed fibres more at the lower margin. Marchand[3] states that the uncrossed fibres were situated more upwards and inwards, the crossed fibres below and inwards—in a case of unilateral optic atrophy.

All previous observations show, that there is considerable difficulty in tracing degeneration beyond the chiasma. In some cases the results seem to point to a mixing of the crossed and uncrossed fibres in the optic tracts. In other cases, the crossed and uncrossed fibres had a more or less isolated course, in distinct bundles, but in none were these bundles sharply separated, and the exact relation of the crossed and uncrossed fibres, has varied somewhat in different cases.

Through the kindness of the late Dr. Ross, some time ago I had the opportunity of making a microscopical examination of the optic tracts and chiasma, in a case of complete unilateral blindness.

The following is an abstract of the notes which I took on the case during life :—

Complete loss of vision, right eye: atrophy of R. optic disc: left eye normal: cardiac dilatation: œdema and ascites. History of sudden onset of blindness in r. eye, after an attack of rheumatism, four years previous to death. Autopsy. Cardiac dilatation: old perihepatitis: cirrhosis of liver; ascites: atrophy of r. optic nerve.

Hannah T., age 56, was admitted as an in-patient at the Manchester Royal Infirmary, under the care of Dr. Ross, on December 30th, 1890.

[1] Purtscher v. *Graefe's Archiv. f. Ophth.*, xxvi., 2, S. 191.
[2] v. Gudden v. *Graefe's Archiv. f. Ophth.*, xxv., 4, S. 237.
[3] Marchand v. *Graefe's Archiv. f. Ophth.*, xxviii., 2, S. 63.

Previous History.—About eight years before admission, the patient suffered from an attack of rheumatic fever. About four years before admission, she had a second attack. One month after her recovery from the second attack of rheumatic fever, she suddenly became blind in the right eye [?embolism of central artery of retina]. At the time of onset of the blindness, she was sitting on the doorstep exposed to the heat of the sun. The right eye suddenly became almost blind, and began to "water;" but there was no pain. Vision became more and more impaired, and in the course of a week, the right eye became totally blind. The left eye was not affected, and there were no other symptoms of lesion of the nervous system.

Soon after the onset of blindness in the right eye, she suffered from bronchitis and pleurisy. In the summer of 1890 she was an in-patient at the Manchester Royal Infirmary, suffering from dilated heart (probably alcoholic).

Present State:—

December 31st, 1890.—Patient is an exceedingly stout woman. Marked orthopnœa. Purpuric spots scattered over the limbs and trunk. Slight œdema of the legs. Marked ascites.

Circulatory System.—Marked dyspnœa and palpitation; pulse 75, irregular in force and rhythm, feeble and easily compressible. Well marked carotid pulsation. Also pulsation in the external jugular veins, which ceases on pressure on the vein just above the clavicle. Heart: apex impulse difficult to localise on account of obesity. On percussion, apex in the sixth space, four inches from the left edge of the sternum; deep cardiac dulness begins at the third left cartilage and superficial dulness at the fourth. Deep dulness extends half-an-inch to the right of the right edge of the sternum. A soft systolic murmur is heard at the apex, also heard at the aortic region.

Respiratory System.—Cough troublesome. Abundant muco-purulent expectoration. Respirations 36 per minute; râles heard all over the chest.

The abdomen is greatly distended with fluid, which prevents the area of the liver dulness from being mapped out.

Urine scanty, high coloured, 1020, acid, no albumen, no sugar.

Eyes.—Slight external strabismus of the right eye. Pupils R>L. No perception of light in the right eye.

Opthalmoscopic Examination.—Media of both eyes normal. *Right eye.*—Well-marked atrophy of the optic disc. Disc of a dead white colour, margins well-defined. No irregularities of the margin. Slight cupping of the disc. Vessels somewhat smaller than those of left fundus (but the difference is only slight). No white lines along the vessels. No other abnormalities detected in the right fundus.

Left eye.—Vision, field of vision, and fundus normal.

No paralysis of ocular muscles.

No signs of any lesion of the nervous system.

Briefly, the subsequent history was as follows:—

The patient's abdomen was tapped on January 3rd, 1891, and a large quantity of ascitic fluid withdrawn. Soon afterwards, œdema appeared in the right arm, and then in the left. The dyspnœa and cyanosis became more marked. The œdema in the legs increased, and fluid soon re-accumulated in the peritoneal cavity. From January 11th to the time of her death on January 23rd, she suffered from considerable pain in the abdomen.

Post-mortem Examination.—Briefly stated the following were the results of the autopsy:—

Abdominal cavity contained a large quantity of yellowish slightly turbid serum. There were a large number of recent peritonitic adhesions.

Heart, greatly enlarged; weight 32 ozs. Cavities much distended, and orifices dilated. With the exception of some very slight irregularities on the margins of the mitral valves, the valves of the heart were normal. Ventricular walls pale, and showed distinct signs of fatty degeneration.

Liver: Capsule of a dead white colour, thick and firm on section. Liver cirrhotic.

Spleen a little enlarged, capsule white and firm. Kidneys congested.

Dura mater adherent to skull cap. The base of the skull and the region of each optic foramen, normal.

The right optic nerve, from the eye-ball to the chiasma, is markedly atrophied; it is much smaller than the left, which is of normal size. At a point eight millimetres in front of the optic chiasma, the greatest diameter of the right nerve is three millimetres, the greatest diameter of the left nerve, at a corresponding point, is five millimetres. The right optic nerve has a greyish colour, the left is of the normal dead white colour. There are no signs of meningitis, tumour, or other abnormality about the right optic foramen. Nothing abnormal in the right orbit, nor in the region of the chiasma, or optic tracts. The orbital portion of both optic nerves, and the posterior half of the right eye-ball, were removed for microscopical examination. To the naked eye, the posterior part of the eye-ball appeared normal. The basal ganglia of the brain, crura, pons, and medulla, with optic tracts and chiasma attached, were separated in one piece from the cerebral and cerebellar hemispheres, and placed in Müller's fluid. The external and internal geniculate bodies, the corpora quadrigemina, optic thalami, internal capsules, occipital lobes, supra marginal convolutions, and all other portions of the cerebral hemispheres, pons, medulla, and cerebellum, were normal to the naked eye.

Microscopical Examination.—After hardening in Müller's fluid, the various parts were embedded in celloidin, and the sections stained according to Weigert's hæmatoxylin method. Sections of the optic nerves were made about one centimetre behind the eye-balls, again midway between the eyeballs and the optic foramina. Also the whole of the nerves, from the optic foramina to the chiasma, were cut into sections. The whole of the chiasma was cut into vertical transverse sections. The whole of the right optic tract, and the greater part of the left tract, were cut into transverse sections.

Optic Nerves.—About one centimetre behind the eye-ball, the sections of the right optic nerve are smaller than those of the left. The right nerve is ovoid in shape, and its greatest diameter 2 mm.; the shortest 1½ mm. (excluding the sheath of the nerve, which is separated from it by a comparatively wide interval). Sections of the left nerve are roughly circular; diameter 3 mm.; the nerve is closely surrounded by its sheath.

In the right nerve, the connective tissue trabeculæ are greatly increased; the nerve fibres are *almost absent;* only a very few minute bundles of fibres remaining near centre of the nerve, but towards the inferior surface (stained black in the Weigert's specimens). The remaining portions of the nerve consist of granular looking connective tissue (stained yellowish brown in the Weigert's specimens), with an occasional nerve fibre (see diagram 1). The nerve is well supplied with large blood vessels the external coats of which show an increased amount of fibrous tissue. Sections of the nerve, midway between the eye-ball and the optic foramen, and also close to the chiasma, present a similar appearance. Immediately in front of the chiasma, at the inferior (basal) part of the right nerve is a small bundle of nerve fibres, running transversely, and parallel to the surface. These fibres are seen in longitudinal section; the bundle is broadest at the inner side of the nerve, and gradually diminishes to the outer side. The above-mentioned minute bundles of nerve-fibres seen in transverse section, near the centre of the nerve in its orbital portion, gradually take up a position towards the outer side of the nerve, close to the chiasma (see diagram 3). The left optic nerve is normal (see diagrams 2 and 4).

Chiasma.—The whole of the chiasma was cut into vertical transverse sections. In the first (most anterior) sections, a very small bundle of nerve fibres was seen at the inferior surface of the *right half*, corresponding to the fibres seen on the inferior surface of the right optic nerve, immediately in front of the chiasma. The fibres were seen in longitudinal or oblique section, and ran parallel to the surface. The bundle was broadest at the median side, and blended here with the nerve fibres of the left half of the chiasma; towards the outer side of the right half of the chiasma the bundle gradually diminished in size. To the outer side of the right half of the chiasma, a very small bundle of nerve fibres was seen in transverse section. The whole of the right half of the chiasma, with the exception of the fibres just mentioned, consisted of connective tissue (similar to that of the right optic nerve), without nerve fibres. The *left half* of the

chiasma consisted of normal nerve fibres, in the first sections (see diagram 5). Very soon, however, a narrow streak of degeneration (connective tissue, from which the nerve fibres had disappeared) was seen at the inner (median) part of the inferior surface of the left half of the chiasma. This streak of degeneration commenced at the middle line, on the inferior surface, and passed outwards and a little upwards (see diagram 6). Passing backwards, the streak of degeneration gradually increased in size (see diagram 7); and then, in the posterior half of the chiasma, gradually diminished. The streak of degeneration was always broadest at the median side, and gradually diminishing towards the outer side of the left half of the chiasma.

In the *right half* of the chiasma, the nerve fibres gradually increased in number on the inferior surface, and the area of degeneration gradually diminished and became limited to superior surface (see diagram 7). After the first few sections, the nerve fibres were seen in transverse section, at the inferior and outer side of the right half of chiasma, whilst those towards the median part were seen in longitudinal and oblique section, passing from the left to the right half.

In a few of the most posterior sections, a band of fine nerve fibres was still seen, running transversely between the two halves of the chiasma; and it was difficult to make out any tracts of degeneration in either half of the chiasma.

Optic Tracts: Naked eye appearance of hardened specimen.—On division of the tracts at right angles to their long axis, the *left* tract was seen to be decidedly smaller than the right. The greatest diameter of the right tract, a short distance behind the chiasma, was 4 mm.; its shortest diameter 2 mm. The greatest diameter of the left tract was about $3\frac{1}{3}$ mm., its shortest $1\frac{1}{2}$ mm. Also, for a short distance behind the chiasma, the inferior (basal) surface of the left tract was more flattened than that of the right, especially at the inner (median) side.

To the naked eye, the right tract (on division at right angles to its long axis), presented a pale coloured area in the centre. This pale area was ovoid in shape, the broad end

being to the median side (see diagram 8). On division of the left tract, no such pale coloured area was found in its centre. The whole of the cut surface of the left tract had a uniform appearance, with the exception of a very slight pale coloured rim just at the outer margin (see diagram 9). The pale area in the centre of the right tract, was seen each time the tract was cut, almost up to the point at which it divides. It may be here mentioned, that both tracts had undergone exactly the same treatment. The whole of the crura, pons, medulla, and basal ganglia, with the tracts and chiasma attached, had been placed together in Müller's fluid, and the tracts were not cut until the specimens were thoroughly hardened.

In sections stained according to Weigert's method, the right tract presented, to the naked eye, a pale brownish yellow, ovoid area in the centre, corresponding to that seen on the cut surface of the tract (see diagram 10). This pale area was present in all the sections, both in thin, as well as in very thick sections. Microscopical examination revealed an ovoid central area (corresponding to that seen by the naked eye), in which there was an excess of connective tissue (stained brownish yellow in the Weigert's specimens). In this area, the nerve fibres were less numerous, and more widely separated than at the periphery of the tract. Also the fibres were very much smaller than those in the peripheral zone (? atrophied fibres). The increased connective tissue in the central area had a granular appearance, and was well supplied with blood vessels. In section stained with aniline blue black, this central area under a low power, appeared slightly darker than the rest of the section. The area in which the connective tissue was increased came nearer the surface of the tract at the median side than elsewhere. Examination of the left optic tract, stained according to Weigert's method, failed to reveal any area of degeneration in its centre. At the centre of the left tract, the nerve fibres were quite as large, and as numerous, as at the periphery; and there was no excess of connective tissue. But at the inner half of the inferior surface, was an area, in which the nerve fibres were exceedingly scanty, and separated by a large amount of connective tissue (see diagram

11*b*). Extending along the inferior surface and outer side of the tract, was a very much narrower zone, in which there was a slight excess of connective tissue. The changes in the tracts just described, were seen in all the sections, from the chiasma almost up to the external geniculate bodies—up to the point at which the fibres separate into two bundles. The central area (in which the connective tissue was increased) was situated, somewhat more to the median side, however, as the right tract approached the external geniculate body; but otherwise the degenerated portion occupied almost the same position in all the sections, from near the chiasma to the most posterior part. The external geniculate bodies, and the anterior and posterior tubercules, of the corpora quadrigemina, appeared normal on section.

Judging from the changes found in the case just recorded, it would appear that the optic nerve-fibres, on crossing at the chiasma pass to the inferior surface of the opposite half. In the tracts, it would appear that the uncrossed fibres take a central position; and that crossed fibres lie at the inner half of the inferior surface, and, perhaps, along the periphery, at the outer half of the inferior surface, and at the outer side of the tract.

There were no signs of degeneration at the upper surface of the tract, on the side opposite to the atrophied optic nerve.

When one remembers, that the optic tracts are not simply composed of crossed and uncrossed fibres, but contain also a large number of fibres, passing from one tract to the other (posterior commissural fibres—Meynert and Gudden), one would not expect the degenerated part in the one tract, to be the EXACT counterpart of that in the other. Still it is somewhat strange, that in the above case and in the other cases recorded, the changes in one tract do not approach more to the counterpart of those in the other.

The facts, however, of the above case seemed worthy of record: that in a case of atrophy of the *right* optic nerve, the following changes were found:—

Chiasma.—Right half of the chiasma degenerated (anteriorly). The degeneration became more and more limited

to the superior surface of the right half, and gradually diminished (posteriorly). In the left half of the chiasma anteriorly, a narrow streak of degeneration was seen, commencing at the inferior surface, near the middle line, and running obliquely outwards and a little upwards. This area of degeneration soon increased in size, and then in the posterior part of the chiasma gradually diminished.

The *left* (crossed) *optic tract* was distinctly smaller than the *right*. Naked eye examination revealed a pale area in the centre of the *right tract*. On microscopical examination, in this central area there was an increase of connective tissue, and a diminution of nerve fibres.

In the *left tract* there was a small area at the inner half of the inferior surface, in which the connective tissue was increased, and the nerve-fibres scanty. Also a similar, but much narrower zone, extending along the periphery at the inferior surface, and at the outer side of the tract.

Description of Diagrams. Sections of Optic Nerves, Chiasma and Tracts, in a case of Unilateral Optic Atrophy.

All the diagrams represent sections, stained according to Weigert's method, except 8 and 9. The dark portions (dots and lines) represent healthy nerve-fibres (stained black). The lighter portions represent degenerated parts (stained brownish yellow). The numbers are placed at the *inferior* surface of the diagram of each section.

1.—Right optic nerve and sheath, 1 cm. behind the eye-ball, × 5 diameters.

2.—Left optic nerve, at a corresponding point, × 5 diameters.

3.—Right optic nerve, immediately in front of the optic chiasma, × 5 diameters.

4.—Left optic nerve, immediately in front of the optic chiasma, × 5 diameters.

5.—Section (transverse vertical) of most anterior part of chiasma, × 5 diameters.

6.—Section of chiasma a little more posteriorly.

7.—Section of chiasma about the centre.

8.—Naked eye appearance of surface of right optic tract, on transverse section, × 2 diameters.

9.—Naked eye appearance of surface of left optic tract on transverse section, × 2 diameters.

10.—Transverse section of right optic tract (Weigert's stain), × 10 diameters; (*a*) = degenerated part.

11.—Transverse section of left optic tract (Weigert's stain), × 10 diameters; (*b*) = degenerated part.

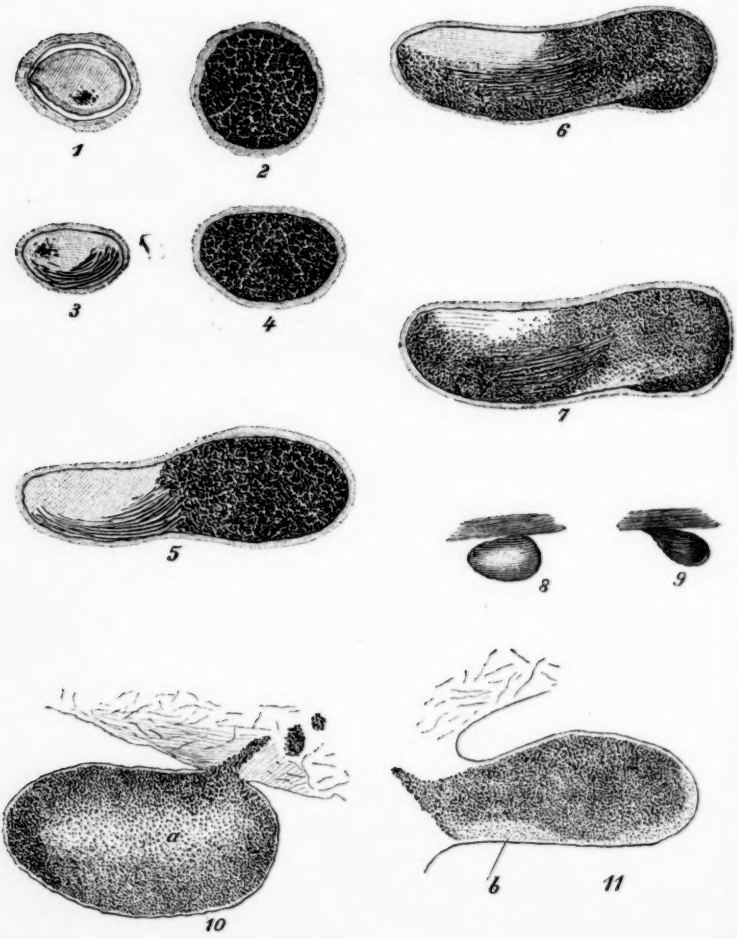


FIG. A. To show obliterated vessels—at *x* and other parts. Transverse section of Ulnar N. at wrist (alum-carmine).

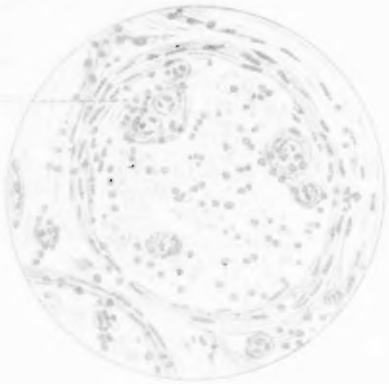
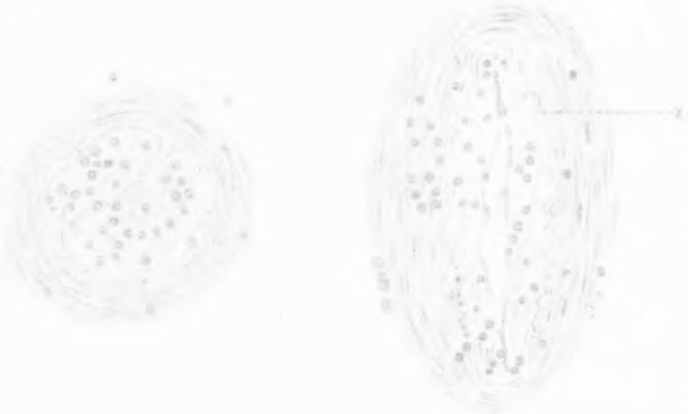


FIG. B. Transverse section of an artery lying outside a funiculus of Median N. at wrist. To show the thickening of internal coat. At *x* the elastic layer of this coat (alum-carmine, × 360).

FIG. C. Transverse section of an artery lying outside a funiculus of Ulnar N. at wrist. To show complete occlusion (alum-carmine, × 480).



ON CERTAIN MICROSCOPICAL CHANGES IN THE NERVES OF THE LIMBS IN CASES OF GENERAL PARALYSIS OF THE INSANE.

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WE examined microscopically nerves from the limbs of ten patients, taken without selection, with the object of ascertaining any morbid condition which might be present. Of these, nine were undoubtedly general paralytics, judged clinically and pathologically; one case did not present the pathological appearances commonly found in the brain and surrounding membranes in general paralysis, but such clinical symptoms as presented themselves were compatible with that disorder, and the peripheral nerves showed in marked degree the changes exhibited by the nerves in the other undoubted cases.

The ages of six of the patients at the time of death ranged between 41 and 49 years; two were 36 and 37 respectively; one 29, and one 58.

The duration of the disease (in years) was, in two cases, 4, in two others 2, in one 6, in another 3. Two others died 18 and 14 months respectively from the time at which symptoms were first observed. Lastly, in two cases symptoms of insanity had been noticed for four months only—this period including observations made outside and inside the asylum; but in these the real duration of disease cannot be even approximately given.

Mention is made of atheroma—existing either in the vessels at the base of the brain or in the aorta—in six cases.

It is noteworthy that no evidence of *syphilis*—either clinical or pathological—was forthcoming in any of the cases. Apart from the examination of the patients, inquiries were made of the relatives, in the usual way; no history of specific disorder was obtained, however: the usual doubt of course attaches to the replies. Nevertheless, the fact is interesting, and, viewed in the light of the morbid change shortly to be described, significant—that no evidence of syphilis was obtainable in any single case.

The nerves examined were the median, ulnar, and posterior tibial, on each side and at the following sites: median and ulnar at the wrist-joint and in the middle of the arm, posterior tibial behind the internal malleolus, a short distance above the point of division. At each site a portion of nerve about 2½in. long was removed; this was hardened in Müller's fluid, and subsequently in a solution of bichromate of potash (four per cent.), or in Ehrlitzki's fluid. Certain of the nerves were hardened and stained in osmic acid (one per cent.) Sections were cut in transverse and longitudinal directions. For staining we commonly employed anilin blue-black (0.25 per cent. aqueous sol.) alone or in combination with hæmatoxylin (the combination being particularly adapted to the staining of axis-cylinders and connective tissue); alum-carmine, and the Weigert-Pal method. Portions of nerves were also cut fresh (æther-frozen) and stained with anilin blue-black and with osmic acid.

Our attention was attracted to the following morbid conditions: (*a*) indications of chronic inflammation and degeneration, (*b*) obliteration of blood-vessels.

(*a*.) These indications were as follows:—(1) With nuclear dyes:

Increase in the amount of connective tissue, especially affecting the perineurium and its prolongations, and the endoneurium. Here and there blocks or masses of connective tissue. Small round cells in numbers throughout the bundles and especially numerous about the blood-vessels; here and there masses composed of such cells. Nuclei prominent and numerous in many bundles.

Fat increased in amount between the funiculi.

Blood-vessels numerous, both without and within the bundles; many of large size, and very many full of corpuscles (the congested vessels are a prominent feature). Thickening of the walls of the vessels, and increase of nuclei upon them.

Alteration of nerve fibres. Marked variation in size was exhibited; by the side of quite small fibres were others, very large and irregular in shape (these were apparently swollen), and often with homogeneous, structureless aspect. Numbers of small fibres also visible. Granules of myelin frequently met with, about small heaps of such granules were collections of round cells. Much variation in the state of the axis-cylinder was observable: the stained area representing it was often of considerable size, whilst the degree of staining was faint; sometimes a still greater increase was apparent, and then the transverse section of the nerve—with the exception of a narrow ring of myelin outside—was represented by a patch, faintly stained, and uniform in tint: an appearance indicative of disease of the axis-cylinder alone, or such combined with morbid change in the quality of the myelin, of a kind permitting of absorption of nuclear dyes. In rare instances no surrounding ring of myelin was visible. In many of the most altered nerves there was no indication of axis-cylinder.

(2) With the Weigert-Pal method:

Whereas the fibres of a healthy nerve are stained by this method with, at any rate, an approach to uniformity, those of the nerves under consideration reacted to it very unequally. Alongside broad, well-stained rings of myelin, enclosing a small unstained area (healthy fibres) were others, narrow, and faintly-stained, limiting an unstained patch of comparatively large diameter. The method brought out also the irregular contour of many of the nerve-fibres, the number of small fibres (in many bundles), and lastly, the granular, broken-up state of the myelin. The sections stained by this method were checked by like ones treated with nuclear dyes, they were also compared with similarly-stained sections from a healthy nerve. Unless such com-

parison be made, misleading results are likely to be obtained, in the case of nerves stained according to the Weigert-Pal process.

To the above summary of the signs of a chronic neuritis[1] a few remarks may be added. These signs were far more marked in what may be called the "low" nerves (those at wrist and ankle) than in the "high" nerves (those in arm); in the latter, indeed, a vascular congestion, with an increase in thickness of the walls of the vessels, and an accumulation of round cells about them, often constituted the most prominent morbid features.

In the case of the "low" nerves, we observed that sections taken from the peripheral extremity of a piece of nerve between two and three inches long not infrequently showed the signs of disease described above in much greater degree than did sections from the central extremity.

Corresponding portions of nerves—whether at arm, wrist or ankle—were found to be very unequally affected.

Lastly, there was by no means a dearth of healthy nerve fibres, notwithstanding that so many fibres showed the morbid conditions above enumerated.

The state of the spinal nerves in cases of general paralysis of the insane has been very little inquired into, so far as we can gather.[2] Déjerine[3] describes morbid appearances which he met with in one case: the myelin and axis-cylinders were considerably affected. Bevan Lewis,[4] many years since, dealt with the histology of the great sciatic nerve in cases of general paralysis.

(*b.*) Obliteration of blood-vessels.

In addition to the vascular changes above described the following morbid appearances pointing, in our opinion, to obliteration of blood-vessels, were present in eight out of ten cases. Patches, circular or oval in shape, varying in size, and composed of organised tissue were seen both within

[1] Present in greater or less degree in nine out of ten cases.

[2] Mendel (*Die progressive Paralyse der Irren*, 1880) refers to the lack of information upon the subject. He himself does not describe any morbid condition in the spinal nerves. We have not met with any more recent publication containing reference to such.

[3] *Archiv. de Physiol.*, 1876.

[4] *West Riding Asylum Reports*, vol. v., 1875.

and without the funiculi—chiefly within, and more particularly in proximity to the perineurial sheath (see illustration A). Stains such as logwood and alum-carmine bring out the connective tissue of which these patches are composed, and often clearly show its concentric arrangement. It should, however, be noted that although the areæ referred to frequently present a "formed" or "organised" appearance, as illustrated in the diagram, they sometimes look homogeneous and almost structureless, being uniformly and faintly stained (in some cases scarcely at all stained) and presenting hardly a sign of their actual composition. One is inclined to suppose that the latter appearances indicate a later stage of disease than the former; that, in fact, they point to a retrograde change in the connective tissue of which the patches are composed.

Apart from the suggestiveness of the appearances just described, reasons may be adduced in favour of the view that they are indicative of an obliteration of the blood-vessels.

1. In many instances the mass of organised tissue has around it a ring, or two or more rings of nuclei having precisely the appearance of the nuclei of muscular fibre-cells, and another outermost covering composed of connective tissue, and having the appearance of an adventitial sheath.

2. The circular patch of connective tissue sometimes lies in a prolongation inwards of the perineurium, next to a small blood-vessel, although the diameter of the former is larger than that of the latter, and the two structures might be considered very different, on superficial consideration, much the same description is applicable to the coverings of the one and the walls of the other. If we suppose that, in the first instance, two vessels of equal size lay next to each other, and that thrombosis took place in one of them, the appearances just described are explained, including the disparity of size, for in the course of thrombosis increase in thickness of the vessel-wall is likely enough to occur.

3. Blood-vessels may be seen in which a clot is surrounded by a thickened *tunica intima:* the lumen is evidently diminished in size. Or there may be considerable thickening

of the internal coat, the opposite sides of the vessel nearly meeting in the middle line (see drawing B). In those instances obliteration appears to be in progress.

4. Structures are visible, composed of a central organised mass and three circularly-disposed coverings (external, connective tissue; middle, muscular; internal, elastic); these are undoubtedly vessels which have undergone obliteration (see illustration C).

5. Between and parallel to the nerve fibres in some *longitudinal* sections capillaries may be seen, presenting at intervals in their course short fusiform dilatations. At these places the lumen of the vessel is obliterated; only a plug of connective tissue presents itself, whereas blood-corpuscles are clearly visible within the vessel at other parts, where its diameter is uniform. (Here and there along the course of the capillary its walls are distinctly thickened, so that the lumen is encroached upon.) It seems probable that the patches under consideration represent transverse sections of these fusiform swellings.

Proceeding, then, upon the view that occlusion of vessels is the morbid condition present, it may be further noted that the vessels chiefly affected are small ones—capillaries, and those a little larger, the walls of which show some muscular fibre. Considerably larger ones may be involved, as the drawing C shows: here the obliterated vessel is seen to have a well-marked internal elastic layer. We were not fortunate enough to obtain a longitudinal section showing plugging of one of these large vessels.

The appearance indicated at x in diagram A is frequently met with; possibly it is due to the implication of two vessels in juxtaposition.

In each of the eight cases which showed the obliteration of vessels the morbid condition was found to be present in about one-half of the nerves examined; thus, out of ten nerves four or five presented it. The "low" nerves were affected in this way far more frequently than the "high" ones. Corresponding parts of corresponding nerves were found to be very unequally implicated: in some instances, in fact, sections taken from a nerve on one side showed no

morbid condition of the vessels, whereas those from the same part (approximately) of the corresponding nerve on the opposite side of the body contained great numbers of obliterated vessels. In a given section the number of funiculi showing plugged vessels varied considerably; thus, in one section, twenty out of twenty-seven funiculi contained them; in another, composed of almost as many bundles, only two contained plugs. The number of plugs contained in one bundle varied from one to ten.

It is noteworthy that whilst the two morbid conditions here described (chronic neuritis and obliteration of vessels) commonly occurred together, several instances were observed in which vessels were obliterated within a funiculus whilst the surrounding nerve-fibres and connective tissue appeared, so far as one could determine, healthy. This fact is incompatible with the view that the obliteration of the vessels is a result of the surrounding inflammation in all cases.

In this connection may be mentioned an explanation of the mode of production of the condition in question put forward by Mendel:[1] according to him the adventitial spaces of the small cerebral vessels are filled with white and red corpuscles and masses of pigment; at some parts the collection is greater, and at these the vessel is compressed to such an extent that the lumen may be obliterated and thrombosis result. In the sections examined by us there was no evidence at all that the plugging of the vessels was due to compression by products of exudation.

Obliteration of the vessels of the cortex cerebri in cases of general paralysis has been described by various writers,[2] but hitherto we have not found any description of or reference to a similar state of the vessels of the peripheral nerves in that disease. In the case of the cerebral vessels, "This obliteration has usually been attributed to proliferation, sclerosis, and contraction of the vessels, or of their outer tunic, or occurring external to it; the newly-formed material constricting the vessels here and there, either occluding them

[1] *Op. cit.*, the Author refers to *cerebral* blood-vessels; those of the peripheral nerves are not mentioned.

[2] See Mickle, "General Paralysis of the Insane," second edition.

directly, or by the intermediation of thrombosis. . . ." (Mickle). In the case of the nerves, although, as said, there was no evidence that the vessels were constricted by newly-formed material, there was no doubt of the fact many occluded vessels passed through tissues affected by chronic inflammation; and the occlusion was possibly due to spread of the morbid process to the vessel-wall. But in several instances, as stated above, vessels completely plugged could be seen lying in healthy-looking tissue. It may be argued that even here the real cause of thrombosis was probably inflammation around the blood-vessel, the changes attending which might have been discovered had the sections been taken from a lower part of the nerve. This theory, however, does not account for the disproportionate degree in which the internal coat is affected, in vessels large enough to show an elastic layer at the outer limit of the *tunica intima* (see the sketch, B). In these the increased thickness of the internal coat is often remarkable, any similar alteration of the other coats being of a subordinate kind. The intima is generally and equally affected; its appearance is that of ordinary connective-tissue (the cellular elements are, however, more prominent than in well-formed connective tissue); there is no evidence of fatty change.

The presence, as in the case of the vessels just referred to, of a line of demarcation between the internal and muscular coats—the elastic layer—renders it easy to localise a morbid change in respect to these coats. But in the case of vessels rather larger in size than capillaries (the actual capillaries need not, for obvious reasons, be considered), with walls composed of internal, thin muscular, and external coats, the degree in which a particular coat participates in a morbid change cannot be stated with any precision, since the coats are not clearly marked off from each other. Many such vessels, with thickened walls and narrowed bores, could be seen in the sections we examined. It may be added that in some instances the narrow lumen was occupied by an irregular mass of deep yellow pigment, everywhere in contact with the irregular inner boundary of the internal coat of the vessel. These conditions probably illustrated early stages

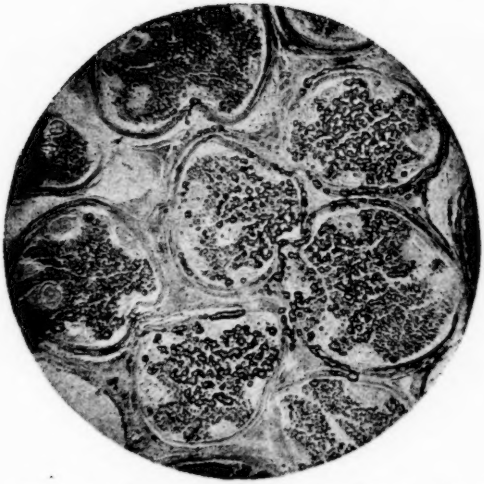

FIG. i. Transverse section of a nerve: low power. The unstained patches within the funiculi indicate plugged vessels.

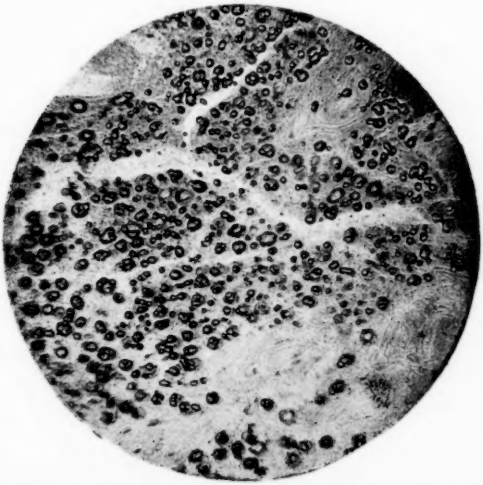

FIG. ii. Part of i. under higher power. Shows degenerated nerve-fibres. The faintly-stained areæ indicate plugged vessels. (Figs. i. and ii., Weigert-Pal stain.)

in the obliterative process, as to the cause of which we are not, in the present paper, disposed to speculate.[1]

Our thanks are due to Dr. C. MacPherson, Wadsley Asylum, for kindly furnishing the drawings illustrating the diseased condition of the blood-vessels.

[1] Since this paper was written we have examined certain nerves in three more cases of general paralysis. In one case the nerves at the wrist showed the changes in the vessels described above; in the other two cases no such changes were found in the nerves examined, but the experience afforded by the cases detailed above justifies us in observing that they may nevertheless have existed in other nerves not examined.

ON HEREDITARY ATAXY, WITH A SERIES OF TWENTY-ONE CASES.

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SINCE Friedreich first described Hereditary Ataxy in 1861, many cases (in all less than a hundred) and series of cases have been reported, which have been supposed to belong to this disease; but in some instances the reports have been so meagre, and in others there has been such wide variations in the symptoms present, that it has so far been well nigh impossible, from the literature of the subject, to obtain a definite idea of the conditions which must be present in any given case, in order to entitle it to a place under this heading.

The establishment of a reliable and permanent criterial picture to be used for purposes of comparison will necessarily require many years for its completion, more especially in a disease so rare as this one. The establishment of such a standard, however, worked out from a critical scrutiny of all available cases, is of the utmost importance, even if it have to undergo considerable amendment from time to time, as the stock of definite facts accumulates.

The work of Dr. P. Ladame in this direction (BRAIN, vol. xii. p. 467) is highly commendable; and to it I would refer anyone who wishes to acquaint himself with a brief historical review of the subject, as well as with the views that many distinguished neurologists have held, and still hold, regarding the disease.

After a critical review of all available published cases, and an elaborate study of a case for some time under his

own observation, which he accepts as typical, Dr. Ladame proceeds to erect a criterial standard which he summarizes as follows:

"Slow and progressive ataxy of the four limbs, usually attacking several children of the same family, dating very often from very early age—commencing in the legs, extending gradually to the trunk and arms, the muscles of the larynx, those of the tongue and eyes; weakness of the legs, increasingly difficult gait; choreiform unsteadiness; static ataxy; difficulty of articulation; nystagmus; spinal curvature; paralytic club-foot; abolition of knee-reflexes; no sensory disturbances; absence of oculo-pupillary anomalies and of lightning pains; integrity of sphincters."

Further on Dr. Ladame states that there are no authentic cases known of onset after the twentieth year, that there is integrity of the special sense organs, and that horizontal nystagmus, static and dynamic, is rarely absent. He also speaks of scoliosis and scanned speech as common.

If the cases herein reported are accepted as instances of this disease, then the criteria as set forth in the above summary should be considerably modified. And the same may be said of the descriptions found in the text books or elsewhere, so far as my reading has extended.

Case XVIII.—A business man, single, of temperate habits, with good family history, excepting that his mother became ataxic at about thirty-three, the disease progressing steadily, till she died of tubercular diarrhœa at forty-eight. Her mother and maternal grandmother were similarly affected, but I will not proceed farther with a verbal description of the heredity, as this may be seen by reference to the accompanying chart.

Patient was always active and vigorous in every way until attacked by his present disease, being rather among the foremost in all athletic sports and school work.

His attention was first attracted to this disease while he was working with a surveying party in Texas, and then it was noticed by others before he himself noticed it. He was then twenty years old, and the ataxia was so marked as manifested by a staggering gait, that his chief thought him intoxicated. But now he recollects that at least two years before this, or when he was eighteen years old, the draughtsmen in the office where he worked complained that he rendered their desks or drawing-boards so

unsteady when he leaned against them that they could not well go on with their work; and he further distinctly remembers that on a certain occasion at about this time, when in company with other young men in the country, he was quite unable to read the large letters of an advertisement at a considerable distance, though each of his companions could read them with ease. He feels confident that this comparative visual defect had not always existed. I should say here that this patient has a good English education and that his intellectual capacity is distinctly above the average, so that, notwithstanding his affliction, he would at the present time be justly regarded as a well-informed man; and in this same connection I wish to say further that I can hardly find words in which to properly express my admiration for the high humanitarian spirit and untiring energy which this gentleman has all along displayed, in assisting me to present to our profession all facts that might throw any light upon this obstinate disease.

He feels quite certain, too, and in this he is corroborated by his older relatives who have had an opportunity of observing him carefully, that at the age of fourteen, when his voice underwent the change incident to puberty, that there appeared a gradually increasing defect in utterance. His speech was slower and his syllables less definite and distinct than formerly. The ataxia has always been much more pronounced if he was fatigued, and he now remembers that at the age of eighteen he could not walk in a straight line when very much tired.

There has been some progressive loss of power in the legs from the first, but this has been insignificant throughout in comparison with the ataxia. The ataxia was first noticed in the legs and has progressed more rapidly in their muscles than in other situations, but it has been distinctly noticeable in the arms from a very early period, and has certainly progressed. An increase of the knee-jerk was probably an early symptom, because it has been so in all the other members of the series whom I have examined (in one, a younger brother of the patient, the disease is just now commencing to develop); and because the patient has shown me notes of his case made four years ago by such competent observers as Dr. Henry M. Lyman and Dr. Walter Hay. The former physician made, I believe, the diagnosis of ataxic paraplegia, which, at the time of his examination, and without the more complete family history since elaborated, was probably more nearly the proper nosological position of the disease than any other according to the standard text-books; and even now I

think hereditary ataxia paraplegia is a designation that comes pretty near the mark in assigning to this series of cases a place in accordance with the usual classification of degenerative diseases of the nervous system.

At no time has there been any pain or any other disturbance of sensation. There has been no atrophy, spasm, or trophic manifestations. The sphincters have not been affected, and the patient thinks sexual power has not been more impaired than can be attributed to the general decline in bodily weight and strength which has slowly supervened within the past six years, the weight having fallen from 136 to 112 lbs. in that period.

I should have stated that a tendency to choking while eating has all along been a very troublesome symptom, and is in some way (whether mainly from inco-ordination or paralysis, I can't say) due to the tendency of food to come into contact with the larynx; and this has been a troublesome symptom in several members of the series.

For several years past vision has progressively failed, so that he could read best in a dim light.

Present Condition.—Patient is considerably emaciated, though he eats fairly and sleeps well. He is of medium size and well formed. The sensibility is normal, the knee-jerk is greatly exaggerated and equal on both sides; there is a slight ankle-clonus, the skin reflexes appear in the main normal, but the cremasteric and abdominal are not strongly pronounced. There is marked ataxia in nearly all voluntary movements; so that the patient can only walk when supported by an attendant, and can only stand when leaning against some solid support. Closure of the eyes does not materially increase his difficulty. The gait is such as would usually be described as cerebellar, the patient leans rather backwards against his attendant and sways from side to side, and he has an uncomfortable sense of insecurity all the time, as if his head must fall violently to the ground. He experiences a distinct loss of power in the legs, which he thinks is even greater than could be accounted for by his general decline in bodily vigour, but the muscles are firm and well developed. All the voluntary muscular movements are slowly performed, and of this the patient is quite conscious. He cannot reach out his hand suddenly to seize any given object. The hand moves slowly and deviates several inches in various directions from the direct line that would normally be taken in such an effort. This tardiness of movement is plainly noticed when the patient raises the eyes; on being addressed or in winking, the eyelids will rise so slowly

as to be suggestive of temporary ptosis, but in the end they are raised too high, so that the sclerotic is often so much exposed as to display an expression, usually associated with some intense emotion, when in fact the patient is suffering from no emotional disturbance whatever. Though ataxia in the muscles that move the tongue would be difficult of conclusive demonstration, because the normal movements could hardly be definitely described (and the same might be said of the muscles that move the lips), yet a careful observer would see at a glance that the movement of both tongue and lips, especially the former in this case (because a full beard is worn), were far wider in range than usual. There is no difficulty in swallowing, excepting the tendency to choking already noticed, which might be explained by ataxia and tardiness of the muscles concerned, because no extraordinary effort has to be made, and there is no tendency for liquids to pass out through the nose.

There is marked inco-ordination of the various muscles of facial expression, which is easily observed when the changes are going on incident to the discussion of an absorbing topic, the action being more marked, now in this group of muscles and now in that, so that people casually meeting the patient frequently get the impression that there is some mental defect.

There is no spontaneous movement during sleep or waking repose, but a comparatively slight voluntary movement gives rise to very extensive and peculiar movements in muscles far removed from those required for the execution of the required act. For instance, where the patient puts out his hand to take a book from a table beside which he may be sitting, the whole upper part of the body goes through a series of irregular movements highly suggestive of chorea. This is particularly the case with the head, which is somewhat inclined forward, moved from side to side, and the chin is protruded; and there is often an associated movement of the other hand. In none of these movements is there ever anything approaching a jerk.

This case and five others of the series was thoroughly examined by Dr. W. F. Montgomery, Professor of Ophthalmology in the Women's Medical College, whose reports I herewith append. And two of the less advanced cases (19 and 20) were minutely examined by Dr. Casey Wood, Professor of Ophthalmology in the Postgraduate Medical School in this city.

Patient states that for ten years past he has suffered from some gradual impairment of vision, and has had double vision at times, of not more than a few days' duration, but not for several

years past. Examination shows marked ptosis when the patient is at rest, but by an effort he can raise the lids, showing sclerotic above the cornea when the eyes are directed in a horizontal plane. There is perfect co-ordination of the ocular muscles except to extreme right, where there is slight lagging of external rectus—not enough, however, for the production of diplopia; the lids and conjunctivæ are normal.

The pupils respond to light but very slowly; and the same is true of accommodation. Dilatation also occurs slowly on stimulation of the skin of the neck.

Vision is 20/200 in either eye. Snellen No. 5 *can* be read, though with difficulty, at 8 inches in an ordinary light, much more easily in a dim light.

The ophthalmoscope shows decided blanching of the optic discs and lessening of the calibre of the arteries, with slight but distinct atrophic changes in the retina.

There are only slight peripheral limitations of the field, and almost complete colour blindness, red only being distinguished with any degree of certainty.

The above description fairly represents the patient's condition, May 2nd, 1891, when I first examined him, and there was no material change until about October 15th, except that under tonic treatment with galvanism—20 to 60 m.a.—to the spine for ten minutes two or three times a week, there was a considerable improvement in the general health with a gain of about 10 lbs. in weight. At the last-named date the patient was attacked with a severe cold, and in a day or two became unable to walk even with assistance, on account of loss of power in the legs, without any alteration of sensation or the reflexes. There was some cough, but the appetite remained good. To-day (November 13th) there is considerable improvement in the legs, but the power is much less than before this attack.

Case XIX.—Sister of the preceding, first consulted me May 5th. She is twenty-six and has been subject to headaches with nausea and vomiting since childhood; she first menstruated at eighteen, but this function has been too infrequent since, often occurring at intervals of from two to four months. Ataxia first showed itself in the legs when she was twenty, but had not invaded the arms, the organs of speech or the ocular muscles to any considerable degree till she was twenty-three. She then began to notice that vision was indistinct in a bright light, and she would move back from the window when reading. The ataxia had advanced so far in the legs that she was obliged to give up dancing.

Present Condition.—Patient is well formed, and of medium size; she has intellectual faculties above the average and a happy and cheerful disposition. She is in a rather reduced state of general health, as manifested by numerous very painful boils about the hips and thighs, which have been appearing in successive crops for the last two or three months.

The cutaneous and muscular reflexes are all distinctly exaggerated, especially the knee-jerk.

There is not and has not been any disturbance of sensation or of the sphincters. She walks well without assistance of an even surface, but deviates considerably from a straight line, no matter how hard she attempts to prevent it. Closure of the eyes does not materially increase the tendency to fall. The same ataxy of the movements of the hands, eyelids and face are present, as in the preceding case, and the same tendency to extensive associated movements, alike in quality but less in degree. There has been no tendency to choking.

The oculists report slight static ptosis with inco-ordinate over-action of the levators. All the movements of the ocular muscles and all the pupillary reflexes are sluggish. All are present, however, and in no case can nystagmus be produced. Vision is 20/80 and Snellen No. 3 is read well enough at 10 inches. Patient reads much better in a dim light. The average expert would pronounce the fundus normal without knowing anything of the condition of vision.

The colour sense is not much affected and there is some peripheral limitation of the field of vision.

The patient states that she is conscious of some difficulty of articulation, but there is no markedly positive defect.

It should be stated that this patient, the only girl in a family of five children, was, before the onset of this disease, very active physically and accustomed to join in the sports and exercises practised by her brothers; and indeed, all the members of this family were very active, so that in athletic contests at school they very frequently carried off the prizes. For this reason it is easier to determine the date of onset of ataxic symptoms than it might be under other circumstances. With the application of the galvanic current twice a week, the occasional exhibition of fractional grain doses of calomel, six week courses of Fowler's solution carried up to six drop doses after meals, and several short courses of simple bitters with port wine, the boils, the headaches and the irregular and painful menstruation have entirely disappeared, and the special symptoms of the disease have certainly not increased.

CASE XX.—Brother of the two former, æt. twenty-four, well developed, cheerful with correct habits, good business man. The disease began, without exciting cause, at age of eighteen. The unsteadiness of gait and difficulty of articulation came on together, and not until two years later was a defect of vision realized. He has carried a cane for the last few months, but can walk fairly well without one, though he deviates considerably from a straight line in walking. The defect in speech is marked, the labials are often defective; yet he whistles without much difficulty. Occasionally the labials are exploded.

The ocular condition is essentially identical with that of the preceding case according to the oculist's report and my own observation, excepting that ptosis is considerably more marked in the left than the right eye.

There appears to be a general relaxation of the facial muscles in repose and even during commonplace conversation, giving the patient a heavy and stolid expression which is quite at variance with the prevailing state of his mind.

He has had galvanism to the spine, as No. 18, and thinks there has been distinct failure in the power of walking in the past three months.

CASE XXI.—There are two other children in the family, twins æt. twenty-one, in one of whom there is distinct exaggeration of the cutaneous reflexes and slowness of speech, but no other indication of the approach of the disease. The remaining twin is entirely free, up to the present time.

In the next family three cases have occurred.

CASE IX.—Is a farmer's wife, aged sixty-seven, of regular habits, who has borne three children. The disease first showed itself at the age of about thirty-five, by ataxy in the gait, and in the hands a year or two later. At the age of about fifty she could walk only with assistance, and at fifty-eight could not pour tea. For several years, at about this time, she went about the house on hands and knees, but for the past four years has been entirely helpless.

Failure in articulation and vision in right eye began to be manifested at about fifty-seven and advanced very rapidly. Vision with the eye first affected was nearly gone in three years, when it began to fail in the other, about the same course being run. No defect of sensation or the sphincters. Memory has failed much within the past four or five years.

Present Condition.—Permanent spastic contraction of the legs, the three being flexed at about a right angle, which can be partly

overcome by force slowly and steadily applied. When the patient attempts to talk the tongue appears to move in every conceivable position without being protruded; the face undergoes various inco-ordinate movements, the head is repeatedly bent forward and moved from side to side, there is protrusion of the chin, and the arms were flexed and moved forward and backward. The movements are highly suggestive of chorea but less rapid. In articulation there is a marked dwelling on the vowel sounds. When I briefly explained the nature of my errand, the patient partly succeeded in saying she was glad to see me, said she had no pain when I forcibly extended her legs, and correctly identified the parts touched when sensibility was being tested. But she said she had only been blind about two months, showing her great failure of memory.

Oculist's Report.—Husband states that patient used to be a great reader and was able to read good print till about nine years ago. At present she can only just distinguish a bright light.

There is almost complete ptosis. If the lids are held open, as is necessary in making an ophthalmoscopic examination, they remain open for a short time and then slowly close. There is scarcely any action of the external ocular muscles, the eyes staring directly forward when the lids are raised. Pupils of medium size and not responsive to light; media clear. The discs present a typical picture of complete atrophy of the optic nerve.

Case X.—Son of No. 9; farmer, age thirty-eight, single, temperate. First noticed disease in legs at age of thirty-one, and in his hands about two years later. Defects in vision and articulation were first noticed at the age of thirty-five, at which time he began to use a cane. Has occasionally had shooting pains in the legs for the past two years, not very severe, however; for the past few months has not been able to retain his urine as usual, and has had to urinate much more frequently than before, having to make great haste to prevent an accident.

Excepting that he still walks with a cane, and that there is more exaggeration of the reflexes, there being well marked foot-clonus on both sides, the objective symptoms in this case are identical with those already described at length as belonging to Case No. 18. There is no alteration in sensations, as might be suggested by the pain and the bladder symptoms. The oculist's report is also practically identical with that of Case No. 18. The general health of this patient was good, though he said he had lost a few pounds in weight.

CASE XI.—Brother of No. 10; died of exhaustion of this disease at age of twenty-six. First symptoms appeared at age of sixteen, as simultaneous ataxy of the legs and eye muscles. He walked with a cane at nineteen, and was helpless at twenty-one. Voice and vision were both somewhat affected at seventeen, but not very markedly so at the time of his death. There was no defect of sensation nor of sphincter action.

Though I had already obtained a pretty full history of the next three cases from their near relatives, I happened to learn that so competent an observer as Dr. Norman Bridge of this city was temporarily living very near them (about 2,000 miles from here), and he examined them and kindly sent me the following notes:

CASE V.—"Patient is sixty-seven years of age. She has a shrunken, cadaveric body, her muscles having undergone extensive atrophic change; the head is in almost constant motion, much like that produced by chorea; this is especially the case when she is talking, moving her hands, or conscious of being observed; the only difference is that the head movements are less regular than in localized chorea. When lying or sitting she can move her legs and even kick, but these voluntary movements are performed with some ataxic irregularity. She is utterly unable to rise from a chair, and has been so for the past year. She moves her hands and arms about freely, but with the shaking, irregular movements peculiar to the ataxic. She cannot, with her eyes closed, accurately touch her nose nor any other desired point on her body. The voice has the same irregularity as the hand movements; it is not a tremor, but a shaking; she does not hesitate in her enunciation, but, so to say, shakes her words out with poor but not abolished labial and palatal action.

"There is no contraction anywhere. The knee-jerk is considerably exaggerated, but there is no foot-clonus. The reflexes are alike on each side. Nystagmus is absent, but the eyesight is so much impaired that she can only see the spots on playing cards (she plays solitaire much of her time). I tried to make an ophthalmoscopic examination, but under great difficulties, so that I do not feel able to say with any degree of positiveness what the condition of the fundus is. Vision is the same in each eye.

"There is no constipation nor vesical symptoms of any sort. Attempts to swallow fluids often provoke coughing and strangling, doubtless from their entrance into the trachea to some extent. She has learned to swallow with great care and deliberation. Her appetite is poor, tongue clean; she insists that her soft

palate is too low, and it is, in fact, relaxed; she insists, also, that she has intense heat in her back, but to my touch it is normal. Pulse 112. The pupils are only very slightly responsive to light."

I have given Dr. Bridge's notes in detail because I thought it important to have an entirely independent statement from so high a source. I will add that the first symptoms in this case appeared, at thirty-five, in the legs, and has progressed quite steadily down to the present condition. There has been no pain or sensory disturbance, unless the intense heat in the back be so regarded.

Case XV.—Son of No. 5; aged forty-nine, also examined by Dr. Norman Bridge, whose notes I quote: "He walks with an ataxic gait, and his upper extremities show to all tests the same condition as the lower. Patient states that the disease first appeared in the lower extremities four years ago, and in the upper a year or two later; says that the uncertainty of gait is much greater sometimes than at others; walks with a cane, and sometimes has great difficulty in rising from his seat. The knee-jerk is enormously exaggerated, and there is a typical ankle-clonus, these conditions being alike on both sides. There is no nystagmus, no contractions, and no bowel nor bladder symptoms.

"The sight of the left eye is abolished, and has been so for the last four or five years. It has slowly declined since he was serving in the army at the age of about twenty. The sight of the right eye is much impaired, but he can read with a good lens, and says that the print then looks correct to him. Careless swallowing of fluids is apt to carry some into trachea and set up coughing. His voice is like that of his mother's, only in a less degree; he says that his voice was normal until a year ago. He has lost 22 lbs. in the last three or four years. Pulse (sitting) is 104. His face has a congenital twist, it is noticeably longer on the left side. He is apathetic. He denies ever having had any pains in the back or extremities."

I will add that there have been no sensory symptoms, and that acute observers among his relatives have assured me that positive signs of ataxia in the gait were present at forty.

Case XVII.—Female, sister of the preceding, was first affected at fourteen and died from exhaustion, extremely emaciated, at twenty-eight. The disease developed rapidly in her case, and early in its course she had severe pains in the hips and legs, and marked paralytic constipation. The voice and vision were much involved.

Case XVI.—Male, aged forty-two, son of No. 5, brother of 15 & 17, has shown some ataxy for the past two or three years; quite obtrusive when tired, but not otherwise. Ataxia of gait the only symptom.

Case VI.—Female, aged sixty-three. First symptoms at forty-five, consisting of ataxy of legs. The course of the disease has been slow. She can still walk on an even surface without assistance, but has to go very slowly and sways about considerably, usually steadying herself by holding on to furniture. The hands are certainly involved, but not greatly, as she can still do needlework and pour tea. Vision is considerably affected, but she can read coarse print in a dim light. Cannot read in a bright light. Speech has become very slow, but is quite distinct. General health fairly good. Other symptoms negative. History given by a relative, himself affected, who has seen her almost daily for years.

Case VII.—Female, died at forty-six of tubercular diarrhœa. First affected, at twenty-eight, with slight ataxy of hands; a few years later it was noticed in the legs, but it progressed very slowly till she was thirty-five when, after child-birth, it advanced more rapidly. With the exception of the last six months, when she became greatly emaciated from chronic diarrhœa, she could walk alone, but would deviate several feet to either side of her line of direction. Until the last two years of life was able to pour tea. Her voice gradually became very slow and somewhat indistinct, but this did not show itself till about the fortieth year and progressed very slowly.

Vision began to be impaired at thirty-six, and by the time she was forty she could only read very large print in dim light; after forty vision failed more rapidly. Other symptoms negative.

Case VIII.—Female, died at thirty-two, of consumption, only slightly affected. Two of her children are living, aged respectively twenty-two and twenty-five, and are healthy.

Case II.—Female, died at about sixty-five. The disease was well developed in all its usual features at fifty, and for the last fifteen years of life the gait showed cerebellar divergence, if I may use that term, to almost the same extent as obtained in No. 7. This statement is the result of a conference among her surviving children and other near relatives, and though I did not attempt to extract from them evidence of earlier symptoms, it is pretty safe to say, judging from the other cases, that they existed at forty.

Case IV.—Male, died at about forty; I could not learn the

cause of death. He was only slightly affected for two or three years preceding death. No further particulars available.

Case XII.—Female, died of pneumonia at thirty-one. Married at twenty-three and had four children. First symptoms appeared at about twenty, consisting of ataxy in the hands and arms, three or four years later the legs became affected. The disease progressed quite rapidly in her case. Was unable to walk without assistance at the age of twenty-eight. No pain at any time. Mind impaired during the last two or three years of life—hebetude mainly.

Case XIII.—Female, aged twenty. First symptoms noticed consisted of ataxy in legs, beginning at fourteen. The arms were involved in a few months, and vision and voice failed rapidly. The voice is described as slow, harsh, and indistinct. There have been darting pains in the legs, but she still walks with a cane. This and the preceding patient live 1000 miles away, and the above incomplete history was obtained by correspondence carried on by a near relative. I hope later to get a more exact report.

The same correspondent informs me that he has learned from a reliable source that No. 12 has a younger child distinctly affected, but has no further particulars.

Case III.—Male, died at fifty-five of pneumonia. First affected at forty, after a fall; was able to walk with a cane at time of death. Arms, voice and vision distinctly, but only slightly, affected.

Case XIV.—Male; died of phthisis at sixteen; affected at eleven; the symptoms began in legs after a fall; he was never able to walk afterwards. Later they appeared in the arms. I have no information as to voice and vision.

Case I.—Female, died at seventy-two. The first symptoms appeared at forty-five, after the patient had sustained a fracture of the hip by a fall. They consisted in marked ataxia in the hands; after injury she was never able to walk without assistance. No more definite or satisfactory account of the symptoms can be obtained. Various relatives who remember her well, and have since seen numerous and well marked instances of this disease, are certain that she was affected with it for many years prior to her death, in the same way as the other advanced cases.

Case XXII.—A girl of eight has ataxia of the legs with spastic symptoms so that she walks on tip-toe. Her symptoms have developed gradually during the last two or three years. I have not seen the case, but my information is reliable, though meagre.

CASE XXIII.—A boy of six, with paralysis of both legs, from anterior polio-myelitis (?), at the age of two.

CASE XXIV.—A boy of thirteen has never learned to walk, though the legs are not stiff or atrophied. He is reported as all right in every other way.

This completes the list so far as my present information extends.

Cases XXIII. and XXIV. are only cited as having a collateral bearing on the subject; and though there is some probability that XXII. and XXIV. are affected with this disease, I could not reasonably insist upon it from my present information.

In Cases I. and IV. the history is meagre, and though the period at which patient No. 1 lived is remote, yet the fact that this series of cases has been confined to families who have always maintained a good position in society, and whose family record has been well kept and can be readily traced, and also the very obtrusive nature of the symptoms, together with the fact that they are in most cases progressive through a variable number of years, warrant a diagnosis of a comparatively meagre history. For my own part I have become intimately acquainted with several branches of the family, and particularly with that branch from which I have had most of my information; and I can vouch for their superior integrity and intelligence.

Four cases—Nos. 18, 19, 20 and 21—have been under my observation steadily for seven months, and Nos. 9 and 10 for three months; and two cases, Nos. 5 and 15, have been well examined by Dr. Norman Bridge.

Taking these cases alone for a text and assuming them to be cases of hereditary ataxy, the following diagnostic criteria might fairly be deduced:

Hereditary ataxy is a disease which may be traced through several—at least four—generations, increasing in extent and intensity as it descends, tending to occur earlier in life and advance more rapidly. It usually attacks several members of the same family. It occurs most frequently between the ages of sixteen and thirty-five, but it may begin as early as eleven and as late as forty-five. It shows no

marked preference for sex, but it descends through females four times as frequently as through males. Atavism rarely occurs. The influence of an exciting cause can rarely be demonstrated, but in some instances a fall or injury has appeared to determine the onset; and any cause like child-bearing or lactation, which very much depresses the vital forces, may produce a rapid advance of all the symptoms. There is always considerable inco-ordination of all the voluntary muscles, and a sluggishness of the movements which they produce when the disease is well established. This is usually noticed first in the muscles of the legs, but in a few months or years extends to the arms, face, eyes, head, and organs of speech. Sometimes it occurs first in the upper extremities, and sometimes in the organs of speech.

The ataxy is often extreme, and the gait devious, the patient deviating several feet on either side of the intended line of progression, before he loses the power of walking. The ataxy is not markedly increased by closure of the eyes. The sense of posture is perfect.

Some weakness of the muscles of the legs, without atrophy, is frequently an advanced symptom, and occasionally there is permanent spastic contractions of the legs. In developed cases there are usually extensive choreiform movements of the head and often of the arms accompanying all voluntary movements. These irregular movements occur in the hands, legs or head whenever it is attempted to maintain either of these parts in a fixed position by a voluntary muscular effort. Movement ceases during sleep. The pulse rate may be increased to 112 in advanced cases or may be normal.[1]

There is usually some degree of static ptosis, with over-action of the levator on looking upward. In rare cases there may be temporary diplopia, in the early stages, due to weakness of the external rectus. There is no nystagmus of any kind.

Atrophy of the optic nerve is a constant and early symptom, and usually progresses slowly with the other

[1] I am inclined to attribute the pulse rate noted by Dr. Bridge to emotional disturbance, because I have made repeated examinations in the six cases under my care and found it only subject to normal variations.

symptoms. Rarely it begins earlier in one eye than the other.

The response of the iris to light and accommodation is sluggish and diminishes with the advance of optic nerve atrophy; when this latter is complete, as may happen in advanced cases, there may be complete internal and external ophthalmoplegia.

There is always marked disturbance of the articulation, probably due to inco-ordination of the muscles concerned, for weakness cannot be demonstrated. In some cases there is a troublesome tendency to strangulation in swallowing liquids, due to their getting into the larynx, but otherwise swallowing is in no way difficult.

Occasionally the sphincters are slightly, but positively, affected, this symptom only appearing in those cases where spontaneous pains in the legs co-existed, having some of the characteristics of those occasioned in locomotor ataxy. Excepting the spontaneous pains already mentioned, there is no disturbance of sensibility. There are no vaso-motor or trophic symptoms, but there is a marked tendency to emaciation; there is no hypertrophy or valvular lesions of the heart.

The knee-jerk is always exaggerated and there is frequently ankle-clonus, and the cutaneous reflexes are also always exaggerated, but to a less degree. The exaggeration of the reflexes is an early symptom, and they often decline considerably when the disease is far advanced.

There is never paralytic club-foot, nor any other deformity excepting rarely permanent spastic contractions of the legs in advanced cases. In none of these cases have the patients ever suffered from rheumatism so far as I can learn.

I wish to repeat that the above summary of the symptomatology of hereditary ataxy is only intended to apply to this particular series of cases; and I have only presented it in this way so that it might be the more easily compared with other series.

I regret that I am unable to show any sections illustrative of the pathological anatomy of the disease. Of the pathology it seems pretty evident that the prominent fea-

tures consist in an extensive degenerative process affecting mainly in the upper motor segment, that is the cortical cells and the fibres extending from them to the cells in the cord and medulla; and that the tissues concerned have derived a deficient vital endowment from the parent; this deficiency manifesting itself as frequently after, as during, the age of development.

The optic nerve atrophy, the occasional spontaneous pains, associated with muscular weakness of the bladder and rectum, together with a tendency to emaciation, suggest that tissues outside of the upper motor segment may be either primarily or secondarily involved.

The integrity of the muscular sense and the peculiar nature, wide extent and extreme degree of the ataxy, together with the state of the reflexes, suggest that the difficulty lies rather in the efferent than in the afferent paths.

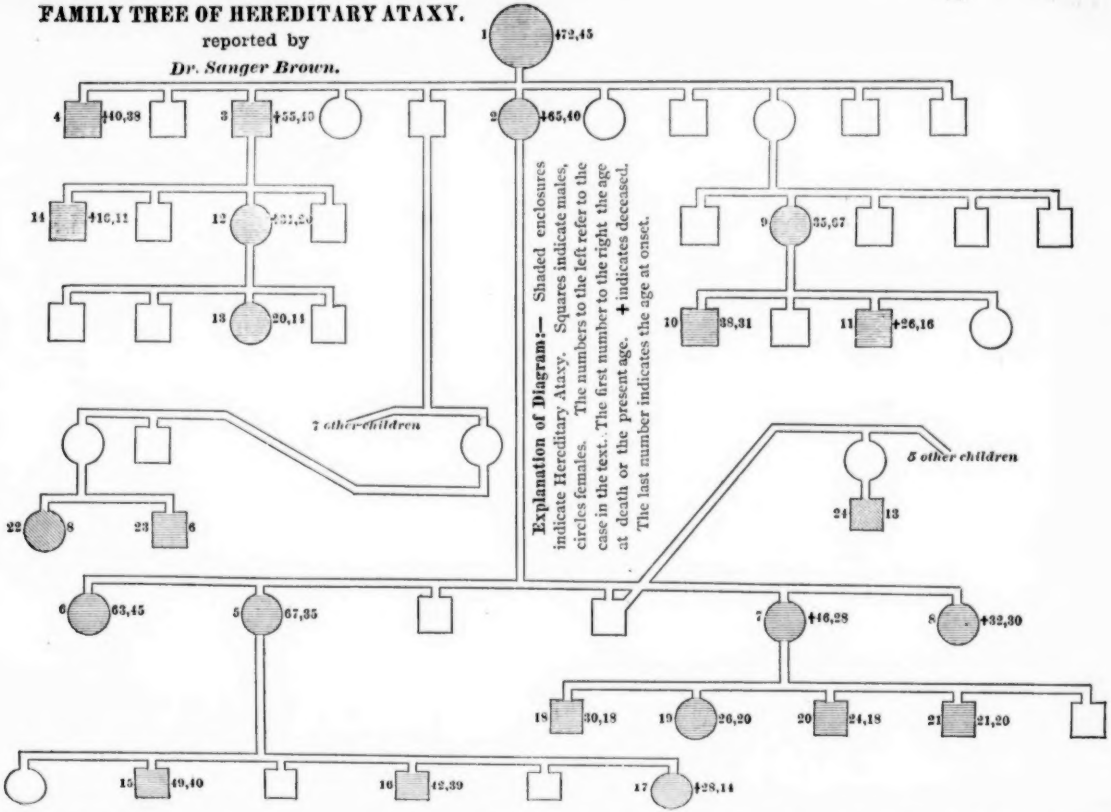
The degeneration is uniformly progressive, though it presents considerable variation in the rate at which it advances in different individuals and at different times in the same individual. I have applied galvanism along the spine in three cases about three times a week for the last five months, using a current varying from 25 to 60 m.a. for ten minutes. The more advanced patient has an impression that the strong current did harm, and he is inclined to attribute the recent considerable loss of power in the legs to that cause; and while such an influence cannot be certainly demonstrated, I should in future cases hesitate to apply a current of more than from 20 to 30 m.a. I changed the poles usually several times during each sitting.

I have paid careful attention to the general health, using wine with a bitter tonic and arsenic from time to time; and all the patients have gained several pounds in body weight. In the case least affected, I cannot observe any advance of the symptoms, but in one there has been an advance of the ataxy noticeable in the gait; and in the third a somewhat rapid onset of weakness in the legs coincident with a bad cold, already mentioned. Now, after about ten weeks, muscular power in the legs has returned to a considerable degree, but not so as to permit the patient to walk.

FAMILY TREE OF HEREDITARY ATAXY.

reported by

Dr. Sanger Brown.



Explanation of Diagram:— Shaded enclosures indicate Hereditary Ataxy. Squares indicate males, circles females. The numbers to the left refer to the case in the text. The first number to the right the age at death or the present age. † indicates deceased. The last number indicates the age at onset.

The main points of difference between the symptoms observed in my series, and the criteria established by Dr. Ladame, I will not occupy space to further indicate, as I have already gone far beyond my intended limits.

With reference to the etiology and symptomatology set down by Dr. Gowers in his text-book, if the age at the time of onset were considerably advanced, and if it were stated that the knee-jerk and skin reflexes might be greatly exaggerated, that in some cases there might be ankle-clonus, and that there might be optic atrophy, the cases herein reported would be fairly well provided for. Indeed, it would seem that there may be much more variation in the age at which the disease first makes its appearance, the distribution and intensity of the pathological process and the consequent symptoms, than has hitherto been suspected.

REMARKS ON DR. SANGER BROWN'S CASES.

I.—BY DR. ORMEROD (LONDON).

Dr. Sanger Brown brings forward a long series of interesting cases, broadly characterised by the title of hereditary ataxy. Ataxia, indeed, was the leading symptom, while the hereditary nature of the disease in the families of this stock is sufficiently obvious. As to the date of onset[1] Dr.

[1] The dates of onset, given in his diagram, fall as follows:—

1st Generation—
Case 1, F, at 45; died at 72

2nd Generation—
Case 4, M, „ 38; „ 40
„ 3, M, „ 40; „ 55
„ 2, F, „ 40; „ 65

3rd Generation—
Case 14, M, „ 11; „ 16
„ 12, F, „ 20; „ 31
„ 6, F, „ 45;
„ 5, F, „ 35;
„ 7, F, „ 28; died at 46
„ 8, F, „ 30; „ 32
„ 9, F, „ 35;

4th Generation—
Case 13, F, at 14;
„ 22, F, doubtful
„ 23, M, „
„ 15, M, at 40;
„ 16, M, „ 39;
„ 17, F, „ 14; died at 28
„ 18, M, „ 18
„ 19, F, „ 20
„ 20, M, „ 18
„ 21, M, „ 20
„ 10, M, „ 31;
„ 11, M, „ 16; died at 26

Sanger Brown says, "it occurs most frequently between the ages of 16 and 35, but it may begin as early as 11 and as late as 45;" as it descends through the family it tends to occur earlier.

As a rule no cause can be assigned for it; but any debilitating influence hurries its progress. This progress is slow in most cases. In two cases, which appear to have been rapid, death took place by exhaustion from the disease in 10 and in 14 years.

The principal symptoms are ataxia of the limbs, usually beginning in the legs and progressing upwards, rendering the patient in time helpless, though muscular power is retained (enfeebled in some instances). With this is associated excess of tendon reactions, often ancle-clonus and sometimes contracture. The skin-reflexes are also exaggerated. No sensory symptoms, except pains in some exceptional cases. As the disease progresses, "extensive choreiform movements of the head, and often of the arms, accompanying all voluntary movements," are seen. "Then irregular movements occur in the hands, legs, or head, whenever it is attempted to maintain either of their parts in a fixed position by voluntary effort."

Articulation is interfered with, apparently by similar kinds of uncontrollable associated muscular action. Sometimes there is a tendency to choke when swallowing. The eye symptoms are peculiar; thus there is a drooping of the eyelids, resembling ptosis, but attempts to raise the eyes may result in overaction of the levators of the lids, giving a staring expression. In one case there appears to have been actual ophthalmoplegia externa. Vision fails, often at an early stage, from a progressive optic atrophy; the pupillary reaction to light is defective, probably on account of this optic atrophy.

Thus it will be seen that Dr. Sanger Brown has described a novel and extremely interesting type of hereditary nervous disease. He himself is inclined to assimilate it to the type known as Friedreich's disease, to which the name of "hereditary ataxia" has hitherto been solely assigned. He holds apparently that the clinical picture of Friedreich's disease

(for which he adopts the excellent description given by Dr. Ladame, in BRAIN (vol. xiii. p. 467) as usually given, should be so modified as to include his new class of cases. To this suggestion we cannot, as yet, assent. Let us first limit ourselves, as the author does, to the discussion of clinical facts. The resemblances between the two types of disease are these:—That they both run in families, and both present spinal symptoms, the main symptom indeed being common to both, viz., ataxia beginning in the legs and progressing upwards, and causing when it first affects the legs an unsteady swerving "cerebellar" gait, rather than the jactitation of the legs which is ascribed to true tabes. With this there is an absence of sensory symptoms, at least in the early stages of both diseases. Such are the main resemblances. Others may perhaps be adduced; first the affection of speech, secondly the irregular "choreiform" movements. It is difficult to pronounce upon either of these symptoms without personally investigating cases; but we suspect from the description (1) that the movements are of an exaggerated character, in comparison with the twitchings and unsteadiness usually witnessed in Friedreich's disease, (2) that the explosive speech differs form the drawling imperfect articulation of Friedreich's disease. Then come in the differences, which seem really to make the clinical pictures very unlike. Thus in Dr. Sanger Brown's cases the tendon-reactions are exaggerated, even from an early stage, and contracture may set in, suggesting, as he rightly says, a primary disease of the lateral columns; whereas in the vast majority of cases of Friedreich's disease the tendon-reaction disappear early, and contracture, though it may occur, is a subsidiary symptom, for (as morbid anatomy has shown) the posterior sclerosis is always more marked and more advanced than the lateral. Those deformities which do commonly occur in Friedreich's disease, viz., lateral curvature of the spine and club-foot, have not been found by Dr. Sanger Brown. The eye symptoms are quite different; thus nystagmus occurs in Friedreich's disease, not in Dr. Sanger Brown's type; and the peculiar action of the levatores palpebrarum which the latter author describes, the ophthalmoplegia and the optic atrophy, are unknown in Friedreich's disease.

On clinical grounds therefore, while we admit the existence of a novel and interesting type of hereditary nervous disease, bearing very important relations to Friedreich's disease, we do not feel justified in identifying the two types. Such identification cannot in fact be made without an appeal to morbid anatomy. Now we know something about the morbid anatomy of the older type, viz., that the nervous centres are found to be smaller than normal, and that there is degeneration of the posterior columns, which in all the *post-mortems* yet made has been of an advanced character, coupled with a less advanced degeneration of the pyramidal tract, and possibly with some other lesions. But Dr. Sanger Brown has not yet had an opportunity of examining any of his cases *post-mortem*.

We will, therefore, collate another set of cases, which appear to bear a close resemblance to Dr. Sanger Brown's; and in one of which a *post-mortem* and elaborate microscopical examination was made. These are published by Dr. M. Nonne, in the *Archiv. für Psychiatrie*, vol. xxii., 1891, p. 283. The accounts relate to three brothers, by name Stübe.

CASE 1.—Heinrich Stübe, æt. 46 at time of examination. (See Genealogical Table, p. 277.)

Was a sailor for fifteen years; was shipwrecked when about 27 years of age, nearly drowned, and exposed for three days and nights. Shortly, but not immediately afterwards, a weakness and uncertainty in the legs began, which progressed, and in two years' time extended to the hands; his speech also becoming affected. In another two years he became practically demented and refractory.

When seen by Dr. Nonne his head was small, forehead low, occiput flat. He walked with crutches (and indeed could hardly stand without them), gait straddling, eyes fixed on ground. Swinging of body when he tries to stand without crutches, not increased by closure of the eyes.

Eyes.—Normal reaction of pupils. Difficulty in ocular movements; downward movements normal, the movements outward, inward, and of convergence, are limited, up-

ward movements still more so. Nystagmus both during movement and during fixation. Optic atrophy moderately advanced, with limitation of field of vision, and impairment of central vision (V.; L. $\frac{5}{10}$).

Facial movements are performed with unnecessary expenditure of energy, and with them are associated abnormal and extraneous movements, *e.g.*, wrinkling of the forehead and eyebrows when he whistles.

Speech immoderately loud, nasal; the words are blurted out.

Upper Limbs.—Slight inco-ordination in fine movements, *e.g.*, in writing. Muscular power normal; sensibility normal in all forms. Tendon reactions normal. No rigidity when passive movements are executed, but after voluntary movements he appears to have some difficulty in relaxing his muscles.

Lower Limbs.—Inco-ordination is manifest when he attempts a given movement, but no loss of muscular power. Knee-jerk lively, but not abnormal; no ankle-clonus. Tendency to "active tension" of muscles as in upper limb.

Mentally, some weakness; thus, he would accuse his brother of cheating and robbing him.

The condition had been stationary during the last few years.

CASE II.—Fritz Stübe, æt. 49 at time of observation.

At 14, noticed an awkwardness of the legs, particularly in going up ladders, walking over planks, &c., and then progressive weakness of the legs, so that at 21 crutches were necessary. Some years later weakness and uncertainty in upper limbs. After some years more, an affection of speech, which rather rapidly reached its present stage. No cause could be assigned for the disease. The symptoms had been stationary for the last ten years.

A somewhat slight and bowed man; head (especially occiput) small, forehead low; mental faculties below par. Stands even more unsteadily than Case I.; unsteadiness not increased by closing the eyes. Manages to walk in a straddling way, by pushing a go-cart in front of him; falls forward without this support.

Eyes.—Some uncertainty in fixing; nystagmus during movement; outward and upward movements limited. Pupils slightly unequal, react to light. Optic discs sharp and pale; field narrowed concentrically to ½ normal; central vision impaired (V.; R. $\frac{1}{6}$, L. $\frac{1}{9}$).

Facial movements exaggerated; wrinkling of forehead, &c., during speech.

Speech loud, nasal, explosive, the breath appearing to be badly regulated (the movements of the uvula, soft palate, &c., appeared in both cases, I. and II. to be normal.)

Upper Limbs.—Inco-ordination in such movements as buttoning, picking up small objects, &c., more marked than in Case I. *Lower Limbs.*—Patellar tendon-reactions lively but normal; skin reflexes feeble; muscular power and sensation normal in all limbs; some prolongation of muscular contraction as in Case I.

CASE III.—August Stübe, æt 40.

From the age of 10, his friends had noticed awk vdness and uncertainty of movement; abnormality of speech as long as he can remember: his tongue "seemed too short;" difficulty in learning.

A thin anæmic man, with slight kyphosis, and signs of chronic pulmonary tuberculosis; mental enfeeblement; head small; low forehead; flat occiput.

Speech (as in last cases) nasal, and showing defective control over the muscles employed; the inspiration and expiration being particularly badly regulated.

Eyes.—Pupils equal, reacting rather slowly; eyes unsteady during fixation and showing nystagmus on movement; upward and outward movements even more limited than in the other cases. No ophthalmic examination was made.

Can only stand with his back against a wall; cannot sit down or rise from sitting without help; can only walk when supported on both sides. When he thus walks, there is no stamping or jactitations of the legs, but a certain unregulated innervation goes on, which calls into action other muscles than those required for the act.

In the upper limbs there was inco-ordination of movement, particularly well seen in the attempt to write.

Tendon reactions feeble in upper limbs; but knee jerk lively. Sensation normal. Difficulty in relaxing muscles after voluntary contraction, but no true contracture.

Evidently, then, these three brothers were affected with the same disease; it attacked the two last mentioned brothers at the age of 14 and 10 respectively, and the other not until he was nearly 30. It ran a slowly progressive course, finally settling down into a stationary condition. The symptoms (to recapitulate briefly) were as follows: progressive ataxia first of the lower then of the upper limbs, without gross loss of muscular power, without contracture (though a peculiar difficulty in relaxing the muscle after voluntary action is mentioned), without affection of the sphincters, without affection of sensation, without material alteration of tendon or skin reflex; peculiar nasal explosive speech, suggesting defective regulation of the vocal cords and respiratory muscles, and accompanied (in the first two cases) by facial movements, over action of the facial muscles during movements of expression, nystagmus, limitation of ocular movements especially in the upward and downward directions, optic atrophy (in two cases). Mental feebleness.

The resemblances between Dr. Sanger Brown's cases and these of Dr. Nonne appear to us very strong indeed, the only differences being that in the second set there were no symptoms of lateral sclerosis (exaggeration of tendon-reaction and contracture); and that there was nystagmus, with perhaps more definite paresis of ocular movement and less of the peculiar over-action of the levatores palpebrarum. Mental degeneration also appears to have been more prominent.

If then we may infer from the symptoms that the disease was in both instances the same, we shall turn with all the more interest to the morbid anatomy of Dr. Nonne's third case. This patient (Case III., August Stübe) died of pneumonia which complicated his previously existing phthisis. The anatomical changes were far less extensive than might have been expected from the symptoms; and may be very shortly summarised under two heads.

(1) Smallness of the nerve-centres—cerebrum, cerebellum, pons, medulla, and cord. (For the exact measurements we must refer to the original paper). This atrophy was unaccompanied by any sign of inflammatory change; and is therefore considered by the author to be primary, and dependent on defective development.

(2) A disproportion in the relative numbers of large and fine nerve-fibres in the spinal nerve-roots. The fine nerve-fibres were greatly in excess (? atrophy or non-development of the large fibres). This anomaly was most marked in the lumbar nerve-roots, and more marked in the anterior than in the posterior nerve-roots.

No degeneration of any spinal tracts was found; indeed the atrophy of the optic nerves (in cases 1 and 2) appeared to constitute the only indication of positive degenerative disease. This therefore constitutes a salient point of difference from Friedreich's disease.

The author does not attempt to explain the symptoms on the basis of the *post-mortem* data; but he remarks that they most nearly resemble those of cerebellar atrophy. The smallness of the nerve-centres is a feature also in Friedreich's disease: indeed Schultze has suggested that it is the primary factor, and that such congenital poverty of development prepares the way for that degeneration of the spinal nerve tracts which has hitherto always been found. (*Post-mortems* upon *early* cases of Friedreich's disease, and upon members of a diseased family who have not yet shewn definite symptoms are much to be desired).

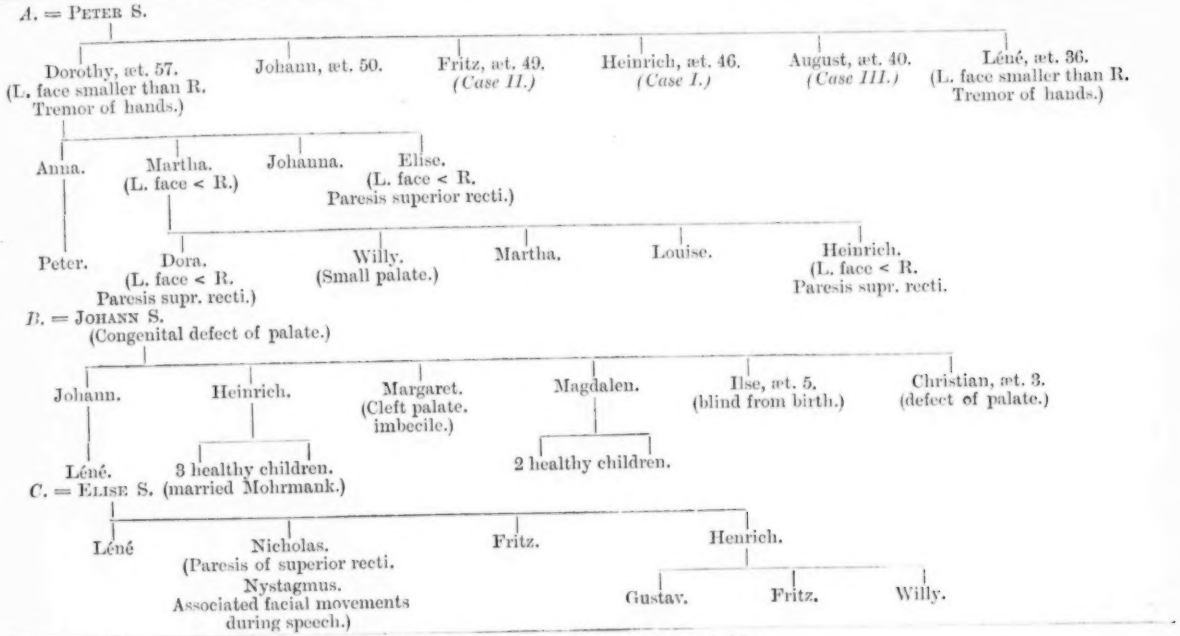
One more case must be briefly abstracted, because it exhibited during life symptoms like those we have already quoted, and *post-mortem* cerebellar atrophy in conjunction with systematic spinal degeneration, resembling that of Friedreich's disease. The account is by Dr. Menzel, *Archiv. für Psychiatrie*, &c., vol. xxii. (1891). The patient had the following family history (we notice only the diseased members). Mother healthy when young; later, developed shaking of the head, and an unsteady gait. Of six brothers and sisters, one brother became affected whilst serving as soldier, with "disease of the spinal cord;"

another was weak in intellect; one sister became affected about the age of 30, she had an awkward gait, and idiotical appearance, and could not hold her head still, committed suicide; another sister had uncertain gait, falling about, and progressive weakness, also committed suicide. Of the patient's seven children, one daughter is stated to have hereditary ataxy. The patient himself, a gold-worker, noticed his handwriting deteriorate from the age of 17, difficulties in walking and in doing things in the dark, from the age of 28 or thereabouts, also some awkwardness in feeding himself, and some pain in the loins, inability to pursue his trade from the age of 40; clumsiness of speech and weakness in right arm from the age of 39. When seen by Dr. Menzel first at the age of 42, head turns to right as he walks (spasm of left sterno-mastoid), facial contortions as he walks or reads: speaks with pauses and effort, as if oppressed or in pain, the longer he speaks the louder and more explosive his words become. All these spasmodic affections are remarkably improved when he lies down. Gait ataxic, with stamping and jactitation of feet, has to hold on to things. Movements of all kinds ataxic, and fine movements impossible. Patellar tendon reactions somewhat exaggerated. No pains. No sensory symptoms. Pupils normal.

At 45 he was wasted, worn, and cachectic. There were now spasmodic movements of the jaw (=opening of the mouth), and involuntary choreiform movements of right arm. Speech still more difficult. At 46, he had difficulty in letting go of things when once he had grasped them. He died about this age. *Post-mortem* there was found (omitting some details) a noticeable atrophy of the cerebellum and pons, atrophy of the olivary bodies, and in the region of the hypo-glossal and facial nuclei. In the cerebellar cortex the nuclear layer was small, and Purkinje's cells very deficient, except in the vermis. The spinal cord was small, and exhibited degeneration of the posterior columns, most extensive in the lumbar and dorsal regions, also degeneration of the crossed pyramidal tracts, and of the cerebellar tracts.

We append Dr. Nonne's tree of the Stube family, which shews that minor degrees of hereditary defect were

We append Dr. Nonne's tree of the Stube family, which shews that minor degrees of hereditary defect were widely spread among the members of it.



A. B. C. in the above table are brothers and sister.

In all the cases then, which we have examined, there was a marked heredity, and a series of symptoms which were both remarkable in themselves, and strikingly similar in the several instances. That these differ largely from those ordinarily observed in Friedreich's disease seems to us obvious. To speculate upon them seems, at present, premature. Nonne's third observation seems to show (what is indeed curious) that they may exist in the presence of simple atrophy of the nerve-centres (and particularly of the cerebellum?) without actual degenerative change. Yet to judge from the facts of Friedreich's disease, where smallness of nerve-centres predisposes to, or at least coexists with, postero-lateral spinal sclerosis, and from the last case (Menzel's) which we have given here, we shall not be surprised to find, in future *post-mortems* on cases of this kind, nervous degenerations as well as want of nervous development. Possibly the want of proper nervous development is the primary and "hereditary" factor, the spinal degeneration a special and engrafted condition, in virtue of which the case becomes relegated to one or other of the types that have been described by Friedreich and the authors quoted in this paper.

II.—BY PROFESSOR BERNHARDT (BERLIN).

It is about thirty years ago that Friedreich described a form of tabes which is developed on the basis of an hereditary, generic, or familial, basis. Erb, in his Handbook of Nervous Diseases, characterises it as follows:—It begins early, between the ages of 12 and 18, perhaps in connection with the change of puberty, or possibly with arrested development of the cord; and affects several, chiefly female, members of a family. Lancinating pains rarely exist at first; but motor troubles are very early pronounced, ataxia rapidly invading the arms, also or even attacking them simultaneously with the legs. Phonatory

inco-ordination, and atactic nystagmus. Sensory symptoms absent, or slight and very late. Cutaneous and muscular sensation long intact. No swaying on closing the eyes. No tendon reactions, bladder trouble, nor decubitus. No psychical disturbance, tremor, nor amaurosis. Paresis, contracture, atrophy, incontinence, appear very late only in this long protracted disorder (over thirty-two years).—Strümpell, in his Manual (Leipzig, 1884) defines the disease in similar terms, and so does Ladame (BRAIN xiii. p. 467), who adds to the list of pathognomonic symptoms, a peculiar club foot and frequent scoliosis. Sanger Brown's very interesting observations show that he had to do with a form of generic ataxy. But it is more than doubtful whether these cases may be classified under Friedreich's type of the disease, inasmuch as they differ from hitherto published instances in many particulars.

(1) Patients suffering from the latter disorder usually fall a prey to it early, between the ages of 12-18. In S. Brown's form this occurs sometimes early, but often much later, *e.g.*, in the forty-fifth year.

(2) Nor does the majority of his patients belong to the female sex; it is only the carrying over of the disease which happen through women.

(3) S. Brown frequently observes ptosis, which is generally absent in Friedreich's form; and universally does not note the occurrence of nystagmus.

(4) Amblyopia and amaurosis, which are never observed in Friedreich's disease, are constant and early symptoms in S. Brown's cases.

(5) In the latter the knee-jerk, which is absent in the former, is increased, and foot-clonus occurs.

(6) S. Brown does not observe club-foot nor scoliosis.

(7) In his cases the pupil-reactions are deficient; a fact not to be wondered at, since in the majority there was amblyopia and amaurosis (optic atrophy); but this symptom does not occur in Friedreich's disease.

S. Brown, at the beginning of his paper, says that if his cases are accepted as instances of this disease, it will be necessary to modify its symptomatology in various

directions; and towards the end returns to this point, on the basis of Gower's description. Erb, however, spoke[1] of two brothers, though there was no nystagmus, and though the knee-jerk was not only present, but lively. Thus whilst authors of repute regard cases as belonging to Friedreich's group, even in the absence of important symptoms, yet we have a number of observations showing us a hereditary ataxy distinct from the Friedreich type, but in several aspects similar to S. Brown's cases. P. Menzel's paper[2] describes the case of a man, 46 years old, with strong neuropathic inheritance on his mother's side. Out of six brothers and sisters three were affected in the same way as himself. The disease began after he was thirty years old; ataxy of the four extremities, rotation of head to the right, with associated movements in the face, speech slow and explosive, large pupils reacting sluggishly to light and accommodation, Romberg's symptom marked, knee jerks exaggerated, but no sensory troubles. Degeneration of posterior columns and roots, as well as atrophy of anterior roots, was found in the lumbar cord. In the dorsal region there was degeneration of the columns of Goll and Burdach, of the lateral pyramidal tracts, and of the lateral cerebellar fasciculi. Similar changes existed in the cervical cord. The bulb showed likewise degenerative changes; the pons was atrophic, and the cerebellum also, where the cells of Purkinje were very poorly developed. Here then we have to deal with defects, rather than with degenerations.

Nonne[3] described a peculiar generic affection of the nerve centres in three brothers with one autopsy. Incoordination began about the thirtieth year; also phonatory disturbances, mental debility, optic atrophy, lively knee jerks; slow progress. The post-mortem evidences were very interesting. The only anomaly consisted in a general uniform smallness of all parts, not resulting from any

[1] Wanderversamml. d. S. W. deutschen Neurologen. (Baden-Baden—June 7 & 8, 1890).

[2] "Beitrage zur Kenntniss der hered. Ataxie," &c. *Arch. f. Psych*, vol. 22, 1891, p. 161.

[3] *Arch. f. Psych*, 1891, vol. 22, p. 283. (See table, p. 277 of the present issue of BRAIN.)

inflammatory process but from a deficient pre-disposition of the nerve centres. There was no trace of the characteristic lesions of Friedreich's disease in the posterior and lateral columns; but as Nonne says, the smallness of the organs was consonant with the anatomical conditions which we are used to find in hereditary ataxy.

Schultze[1] specially called attention to the possibility of the smallness of the cord partly producing the symptoms; and Nonne (p. 314) says: The diseased condition of the optic nerve displays, as a delicate test, that there was in the case a predisposition to degenerative changes, which, however, had hitherto worked no real destruction among all other nervous structures.

I regret that pressure of work does not allow me to go more fully into these interesting questions, but from these short remarks it will appear that the apparently definite type of Friedreich's disease has been modified in various ways. In a strictly systematic sense, as the author himself allows, S. Brown's cases do not belong to Friedreich's type of disease. But if we drop this appellation, and are satisfied with such a name as hereditary, familial, or generic, ataxy, then those cases certainly fall under this interesting category, and bear certain analogies to Nonne's observations.

On anatomical grounds all these cases are characterised by a deficient development of the nerve centres, as compared to their evolution in healthy persons of similar age. This deficiency affects the cord as well as the encephalon (especially the pons, bulb and cerebellum); and in Nonne's case the peripheral nerves also. Post-mortem there may be found nothing but this abnormal smallness, but atrophies and degenerations may also occur. According to the localisation of the latter, variations will occur in single cases, or categories of cases, as to the prevalance of certain symptoms (knee-jerk, optic atrophy, oculo-muscular affections, regional muscular atrophies). The age at which the symptoms begin to manifest themselves will likewise vary from childhood or puberty to riper manhood.

[1] Virchow's "Archiv," vol. lxxix, part 1.

Whilst in one of the large groups of generic central nerve diseases, inco-ordination or ataxy plays the chief part and characterises the affection, there is another such group in which spastic elements predominate ;[1] and yet a third where spinal muscular atrophies and paralyses hold the field.[2]

But all these morbid types, and probably also the other familial nervous affections, may perhaps, as far as autopsies go, be referred to a more or less faulty development of the centres. These defects may prevent the increased demands of advancing years upon sensation and action, from being duly responded to ; or may render the structures more vulnerable to external influences than those of the normally-developed and better-resisting individual, thus giving rise to the characteristic lesions.

[1] Bernhardt ; Virchow's " Archiv." vol. cxxvi., 1891.
[2] Bernhardt ; Virchow's " Archiv." vol. cxv., 1889.

Reviews and Notices of Books.

I.—*Il Cervelleto; nuovi studi di fisiologia normale e pathologica.* Per LUIGI LUCIANI. Firenze, 1891.

II.—*Sull 'origine e decorso dei peduncoli cerebellari e sui loro rapporti cogli altri centri nervosi.* Per DR. VITT. MARCHI. Firenze, 1891.

THESE two works, which are the most recent treatises on the anatomical connections and physiology of the cerebellum, form an important contribution from the modern Italian scientific school. The former gives expression to Luciani's matured views on the subject of cerebellar physiology, and includes the experiments which were recorded by him in the *Prima Memoria* in 1884. The latter records the degenerative changes in the central nervous system of the animals on which partial or complete cerebellar extirpation had been performed. It is our intention here to review in detail both these works, giving special prominence to the former, as it forms a very valuable addition to the physiology of the nervous system, and introduces a new doctrine of the functions of the cerebellum.

As the author has been very successful, both as to the number of animals (dogs and monkeys) on which he has operated, and in the completeness of the partial and total extirpation of the cerebellum, his method of operation deserves mention. Narcosis was obtained in dogs by hypodermic injection of morphia and chloral (morphia 2.5 centigram, chloral 1 gram), and in monkeys by chloroform, in addition to morphia. The muscles were separated down the middle line of the neck, and partly detached from the linea semi-circularis. An opening was then made with a small trephine in the occipital bone, and enlarged by the bone-forceps. The opening thus made extended from the external occipital protuberance to the foramen magnum, so as to expose the whole region as far as the calamus scriptorius. The dura mater was

incised over the middle lobe. *The extirpation of the middle lobe of the cerebellum* is a relatively easy operation. This lobe is separated from the lateral lobes by two antero-posterior incisions. A spoon with a cutting edge is introduced, and the brain matter removed as far as the corpora quadrigemina. When thoroughly extirpated, the floor of the fourth ventricle and the opening of the aqueduct of Sylvius are exposed. To remove *the whole cerebellum* at one sitting, proceed as above described, for the removal of the middle lobe, and then remove the lateral lobes as much as possible *en masse*. The removal of the flocculus, although not impossible, is difficult and uncertain. *One half of the cerebellum* is extirpated by dividing the vermis in the median plane, by means of a Græfe's knife. It is essential that the remaining half be not injured. It is difficult to perform hemi-extirpation of the uvula without at the same time injuring the medulla oblongata. Hæmorrhage from the brain is arrested by means of small sponges, soaked in corrosive sublimate (1 in 1,000).

The disadvantage of operating on monkeys is that the head requires to be held forward by an assistant. In favour of the use of these animals is the very ready manner in which they are narcotised, and the very slight tendency to epilepsy after the operation. In dogs this tendency is combated by hypodermic injections of chloral. The latter, also, are subject to impulsive movements after the operation, and have sometimes to be artificially fed. In monkeys, on the other hand, the irritative phenomena are less intense and more fleeting.

Having overcome the difficulty of removing the cerebellum, we must next see how best to define the phenomena which result from it. In studying the movement of an animal, several principal factors are distinguished—the energy employed in the contraction of the muscles, the tension or tonicity of the muscles, the manner of effecting the contraction, the form of the movements, their symmetry, and their co-ordination, or the manner of association, and succession of movements.

For estimating the force of muscular contraction, the author has made use of a modified dynamometer. He noticed that when a monkey was raised from the ground by means of a belt round the abdomen, it endeavoured to catch hold of any fixed object below it. He therefore attached the hook of a dynamometer to the animal's girdle-strap, raised it from the ground, and allowed it to approach a wooden cylinder maintained in a vertical position by a heavy base. To this the monkey clung with both its hands, and gradually drew out the dynamometer ring, thus enabling the

observer to read on the scale how many kilogrammes it had overcome with the arm muscles. In order to complete and control the observations on cerebellar ataxy, especially as regards the form, symmetry, and co-ordination of movement, he devised a method of tracing the footsteps of dogs on the pavement, by dipping their feet into different-coloured solutions. Throughout the work there are charts, showing the normal impress of dogs' feet when walking and when galloping, and also of those animals, at various periods after the operation, whose cerebellum had been, in whole or in part, removed.

He has studied the nature and origin of the changes consequent on mutilation of the cerebellum under five headings, and they are given here briefly to facilitate the description of the experiments to be presently noted. The theories regarding their causation are detailed later. The effects to be noted are:—

1. *Phenomena of Irritation.* These are dependent on alteration of the functions of the nervous centres provoked by the operation, and are characteristic of the first period, but it is impossible to draw a hard and fast line between these phenomena and those of later dates. 2. *Phenomena of deficiency*, depending upon deficient, unequal, or faulty cerebellar innervation. The symptoms of cerebellar deficiency are: a want of energy for voluntary acts—*asthenia;* a diminution in the muscular tonicity—*atonia;* and the presence of tremors, oscillations, titubation, uncertainty of movement, &c., or *astasia*. Here, again, one cannot separate these from the next group. 3. *The Phenomena of Compensation.* These are determined by those portions of the cerebellum which have not been injured by the operation. Two kinds of compensatory phenomena are observed: organic and functional, the former consisting of the gradual attenuation of the phenomena of deficiency, the latter, of abnormal movements, directed to correct those brought about by the deficiency. The first form is mainly observed in partial extirpation, the second in complete removal, from which we may conclude that other encephalic centres are capable of taking on the functions of the cerebellum. 4. The *phenomena of degeneration* following cerebellar extirpation are detailed when the second work is under review. 5. The *trophic phenomena* show that the cells of the cerebellum exert an influence on the general nutrition of the tissues.

§I. *Effect of division of the cerebellum into two halves.* In three animals on which this operation was performed, one dog lived twenty-two months and from it the observations are taken. There was a characteristic absence of irritative phenomena; there

was indeed tonic extension of the fore limbs, but the *post-mortem* revealed an inflammatory state of the membranes. There was, however, some difficulty in co-ordination. The phenomena of deficiency were represented by a want of energy to perform voluntary acts and a diminution in muscular tonicity and firmness. Tracings showed that compensation went on so well that in course of time it was difficult to discern anything abnormal in the gait. The author concludes that the cerebellum is not an organ which can be divided into two halves without seriously interfering with its physiological activity. It is physiologically as well as anatomically one organ.

§II. *Effects of destruction of the middle lobe.*—This lobe has generally been regarded as the most important part of the organ, especially by Nothnagel, who looked upon the symptoms of cerebellar ataxy as due to direct or indirect lesion of the worm. Vulpian's view was entirely opposed to this: "A section of the cerebellum made exactly in the median plane does not produce the same disorders of movement as lesions a little less deep, but more extensive and affecting one half of the organ more than the other." Luciani destroyed the middle lobe in four dogs and two monkeys. As an example of the effects of destruction of this lobe, I detail the observations made upon a monkey:

May 31*st*, 1884.—After the operation tonic contraction of the muscles of the neck was observed and tonic flexion of the upper and lower limbs. When placed in a hammock the monkey lay quiet in the supine position.

June 1*st*.—When offered a piece of fruit, it stretched out a tremulous arm and took hold of it, but with little energy. Placed by itself on the floor, it was unable to maintain its feet. It tended to fall from right to left.

June 2*nd*.—Arm still tremulous when offered food. It is apparently unable to extend the hand at the wrist, owing to tonic contraction of the flexors. When placed on a table the monkey remained with its limbs flexed, its trunk and neck continually oscillating.

June 9*th*-11*th*.—Gradual improvement in mode of walking. It proceeds with characteristic slowness, but with fair co-ordination of movements. There is still some oscillation on isolated movements. In order to test the difference of muscular energy between the arms of this animal and a healty one, a dynamometer (as before described) was used. The monkey deprived of the middle cerebellar lobe registered 3 kilos., the healthy monkey 8 to 10 kilos

July.—The phenomena are gradually disappearing. There is still some tremor and uncertainty on isolated movements. The dynamometer registers 5.5 kilos. When offered fruit it extends its right hand, tending, however, to lay hold of some article of support with the left.

August.—All the phenomena have diminished to such an extent that there is some difficulty in distinguishing the monkey from a healthy animal.

June, 1885.—The monkey is in good health. Slight oscillatory movements are occasionally observed, which are increased when the animal is frightened. The fore limbs show a force of 9 kilos.

The phenomena observed after extirpation of the middle lobe in dogs, presented little or no variation from those observed in monkeys, with the important exceptions that in place of flexion of the fore limbs tonic extension was observed, while the general agitation immediately following the operation was considerably greater. The general features of the result of extirpation of the middle lobe are: (1) general agitation, tonic extension or flexion of the fore limbs, spasm of the neck muscles and strabismus; lasting from seven to eight days. (2) Muscular asthenia, atonia, and astasia. These are especially observed in the muscles connected with the hind limbs and are of longer duration than the irritative symptoms; they last from ten to fifteen days. (3) The phenomena of compensation ensued in all cases.

It was observed that if the middle lobe was removed perfectly symmetrically, the phenomena of irritation and deficiency were symmetrical; while if one or other side showed accentuated symptoms, it was found that the lateral lobe was injured on the corresponding side.

§III. *The effects of incomplete unilateral extirpation*, that is, extirpation of the right or left lateral lobe. The symptoms observed after this operation, which was performed entirely upon dogs, differed from those following extirpation of the middle lobe in some important points, although the general features were similar. *The irritative phenomena* consisted of curvation of the vertebral axis towards the wounded side, of tonic extension of the anterior limbs upon the same side, of rotation around the longitudinal axis from the wounded towards the healthy side and of strabismus in the same direction. The phenomena of deficiency consisted of muscular asthenia, atonia and astasia on the side of the lesion; while the phenomena of compensation took place gradually leading to restitution of the equilibrium and

ability to walk. A temporary glycosuria was observed in several of these experiments.

§IV. *Effect of removal of one half of the cerebellum.*—This operation was performed upon dogs and monkeys; the effects upon a monkey are here given in some detail:

June 26th, 1884.—Removal of the right half of the cerebellum. After recovery from the narcotism, the monkey presented curvation of the neck towards the right, the head being rotated however towards the left. When placed in a hammock there were movements of rotation around its long axis from right to left, otherwise the animal remains quiet and breathes regularly.

June 27th.—Still a tendency to rotate from right to left, but the animal endeavours to avoid this by laying hold of the network with its left arm. The head and neck are still maintained in the same position as before. The upper and lower limbs on the right side are kept in a position of flexion. The left pupil is more dilated than the right, strabismus of the left eye inward and upwards and nystagmus of both eyes.

June 28th.—Phenomena persist except that the pupils are of more equal size, and there is less rotatory tendency. When attempting to take food there is great oscillation of the right fore limb.

June 29th.—The same phenomena are present, but to a less degree. There is tonic flexion of the right fore limb, but the right leg is relaxed. The impulsion to rotation has almost ceased.

July 1.—Considerable lessening of the symptoms; when presented with a pear, it takes it in the left hand and eats it readily. It uses the left limbs, the right remaining apparently inactive. There are still oscillations of the eyes and body.

Mid-July.—Considerable improvement; it habitually embraces a small wooden column, to which it is attached by a chain, in this way maintaining a vertical position by resting on its ischial callosities. It is beginning to use the right arm more freely. The force of traction of the fore limbs, estimated by the dynamometer, is 3.5 kilos.

September.—Very great improvement; traction by the fore limbs is estimated at 8.2 kilos. When offered food, the monkey holds to the floor with the right arm, while it extends the left. Continuous and increasing capacity for walking; it raises its limbs high off the ground, but the movements are well co-ordinated. The head is still inclined towards the right, and there still remains some strabismus.

April, 1885.—It is somewhat timorous and suspicious in character; the head is still inclined to the right; when walking

the axis of the body is turned towards the right, and planting its right limbs with great abduction, a movement obliquely forwards and towards the right results. When compared with a healthy monkey, a certain indecision in its movements is observed. When offered food it invariably extends the *left hand* to take it. The pupils are equal and the nystagmus has passed off. There is still a slight strabismus shown by deviation inwards of the right eye, and upwards and outwards of the left. A slight ptosis of the right upper lid is also observed.

The symptoms of unilateral extirpation of the cerebellum therefore may be noted as follows:—1. *Phenomena of irritation* are rotation around the long axis from the wounded to the healthy side, associated with strabismus and nystagmus, curvation of the body towards the *wounded* side, tonic extension or flexion of the fore limbs and sometimes of the hind limbs. In two of the monkeys there was temporary glycosuria and polyuria. 2. The *Phenomena of deficiency* are asthenia, atonia, and astasia of the muscles on the side of the lesion. 3. *Compensatory phenomena* were observed in all cases, but a residuum of the symptoms of deficiency existed, and this is readily understood when one remembers that half the cerebellum, corresponding to one half the body, was removed, thus demonstrating that the remaining half is unable to take up entirely the function of the other.

Destruction of one lateral lobe was carried out in dogs and monkeys, which had already lost the middle lobe. The effects did not differ essentially from those which have just been recorded after unilateral extirpation.

IV. *Effects of complete and incomplete extirpation of the whole cerebellum.*—This operation was performed both in dogs and monkeys; one of the latter is given here as an example of the symptoms produced by this mutilation.

June 12, 1886.—Tonic flexion of both fore limbs, the right rather more than the left, slight convergent strabismus of both eyes. It is unable to hold itself upright. About an hour after the operation it was able to approach its back to the corner of the box which was used as a lair, and to take with its left hand a piece of fruit.

June 13.—It rests its back upright in the corner of the box and extends both hands when offered fruit. When relieved of this support the monkey falls and is unable to raise itself up again. There are oscillations of the head.

June 14.—It takes food when resting on the ischial callosities and leaning in the corner of the box. When unsup-

ported has a tendency to rotate from right to left. It can climb up the side of the cage, but cannot remain there without support.

June 16.—Placed on the floor, free from support, it takes a few steps forward, but soon falls to the right. When extending its arm to take the food offered it, it presented an uncertainty of movement not previously observed.

End of June.—The monkey now walks with greater agility, raising its fore limbs high and planting them on the floor with more abduction. There is still a tendency to fall to the right.

July.—As the monkey sits its head and trunk oscillate, but there is less tendency to fall. When eating there is continuous tremor of the arms and trunk. It walks with a hesitating oscillatory gait in a zigzag direction, tending somewhat to go towards the right. It walks more slowly than a normal monkey. During this period the general state of nutrition had somewhat deteriorated.

August.—The ataxic phenomena of the previous month have not entirely disappeared. Its general health is getting worse.

October, 1886—*June*, 1887.—During this period there was practically no change in the phenomena already noted. If anything, there was a slight improvement in its health.

July, 1887.—Is in a good state of health, very little change in the ataxic symptoms. When eating the monkey maintains its position by supporting itself on three limbs, using the fourth to hold its food. When resting in the ordinary way, on its ischial callosities, the hind limbs are abducted so as to enlarge its base of support. It walks inelegantly, with continual oscillations of the head, placing its limbs far apart. It proceeds somewhat obliquely with the axis of the body in the direction of walking. It descends a ladder slowly and with care.

December.—Remains the same as in the July note.

January, 1888.—The animal was killed on the thirteenth of this month. The autopsy showed that the whole cerebellum had been removed with the exception of a small portion of each flocculus, the portion on the left side being larger than that on the right. The cerebrum presented a normal appearance.

The phenomena of irritation following extirpation of the cerebellum do not differ essentially from those following removal of the middle lobe except in their intensity, persistence and greater diffusion. The phenomena of deficiency also only differ in their intensity and duration. The phenomena of functional compensation are more exaggerated.

§ VI. *Effects of extirpation of the cerebellum combined with uni- or bilateral destruction of the sigmoid gyrus.*—It seemed probable

that the compensatory movements arose in the motor region of the cerebral cortex; operations were therefore performed on the sigmoid gyrus. The operations were made entirely upon dogs, and the following is an abstract of the results obtained:—[1]

The first operation consisted of removal of the right half of the cerebellum, and the symptoms were those described in section 4. The second operation consisted of incomplete and asymmetrical destruction of both sigmoid gyri. As a result there was paresis of the four extremities, associated with defective sensibility. The third operation consisted of removal of the remaining half of the cerebellum. The phenomena of irritation were shown in tonic extension of the fore limbs, curvation to the left of the vertebral axis, indicating that the extirpation was complete on the left side and incomplete on the right (and at the *post-mortem* this was shown to be the case). The phenomena of deficiency were marked and persistent; eleven months after the last operation the dog was unable to walk without support. The conclusion to which the author comes at the end of the chapter on this subject may be expressed in his own words:—"The compensatory movements provided for those animals which have been deprived of their cerebellum, and which have become capable of maintaining their equilibrium in the erect posture, when walking and when swimming, are dependent on the sensori-motor region of the cerebrum and they can be suppressed by simple ablation of the sigmoid gyri, which represent the most important part of that region."

Two long chapters are now devoted to an explanation of the phenomena which we have just detailed. An analysis is first made of the irritative phenomena. The "bilaterality" and symmetry of the phenomena depend on bilateral and symmetrical destruction of the cerebellum. In bilateral but asymmetrical destruction or in unilateral ablation they prevail in the muscles on the side exclusively or most effectually destroyed. It is evident that these effects depend on the irritation of the efferent fibres which compose the cerebellar peduncles.

The *irritative phenomena* are: (*a*) pleurosthotonos, or curvation of the vertebral axis towards the wounded side, associated with tonic extension of the fore-limb on the same side, and clonic movements of the other three limbs; (*b*) contortion of the vertebral axis in the cervical region, whereby the head is turned towards the unaffected side; (*c*) nystagmus and strabismus, *i.e.*, deviation downwards and inwards of the eye on the side of

[1] The dog referred to here was shown at the Congress in Voghera, in 1883.

operation, and outwards and upwards on the sound side; (*d*) a tendency to rotation around the longitudinal axis following the direction of the contortion of the strabismus, *i.e.*, from the operated towards the sound side. These are the effects of acute irritation of the peduncular fibres on the wounded side. They are not continuous or inherent after the first or second day; but when the animal is approached, or taken up from its lair, or when it wishes to accomplish movements of defence, or to raise itself, or walk, these symptoms are produced. On the other hand, they become less if the animal is left undisturbed. The acute stage of the irritative symptoms gradually passes away, the tonic spasms losing their intensity and duration, and being transformed into clonic and oscillatory movements. The tendency to rotate and to fall backwards ceases, and the erect posture is possible after a short time. The capacity for walking is always re-established, but it is never possible to say that the irritative phenomena have altogether ceased. In association with the capacity for walking the animal re-acquires the attitude for floating and swimming. In monkeys, as has been shown, instead of tonic extension of the limbs, the characteristic attitude is that of tonic flexion, and further, in these animals, the symptoms are much less intense, and less prolonged, so much so that after a few days the phenomena of deficiency are most marked, every trace of irritation having disappeared.

Of the irritative phenomena one requires special consideration, viz., *rotation around the longitudinal axis*. This had been observed and noticed by many experimenters, and had received various explanations at their hands. The researches of Luciani show that the rotation towards the sound side depends on the complete and successful amputation of the peduncles, the phenomenon being fleeting or partly absent when these fibres are partly untouched. It is essentially a symptom of irritation transmitted to the nervous system by the efferent fibres of the cerebellar peduncles on the wounded side.

Reference is made to two kinds of rotation: (*a*) irritative rotation, caused by traumatic or inflammatory conditions of the peduncular fibres, consists in rotation from the wounded towards the sound side, for in this instance the want of equilibrium is generated by a unilateral exaggeration of cerebellar influences transmitted by the irritated peduncle; (*b*) paralytic rotation, caused by the simple section of a peduncular strand, consists in rotation from the sound towards the wounded side, because here the want of equilibrium arises from the fact that one half of the

centres in connection with the incised peduncle suddenly lose their cerebellar influence, whilst the others retain it. But there appears also to be a form of *vertiginous impulse* which determines rotation, and this was best observed in apes after hemi-extirpation of the cerebellum. These animals learn very soon to avoid rotation, by holding on to the sides of their cage. They cling tenaciously to everything, because the fear of rotation and of injuring themselves conquers their natural timidity, owing to which, normally, they do not allow one to approach them. When placed on the floor they cling to the nearest piece of furniture, and to avoid rotation, rest on the floor not only with their body, but with their head; but after more experience they have recourse to mechanical means to counteract this tendency; that is to say, they rest on the floor with the fore-limbs spread out in abduction, maintaining themselves unmoved for an indefinite time. Anyone who watches these phenomena undoubtedly receives the impression that the monkey suffers from vertigo, and seeks in every way to avoid its ill-effects.

The phenomena of irritation having passed away, the true character of the effects of cerebellar deficiency is revealed. One important fact, which is pointed out by these experiments, is that the phenomena of deficiency are exclusively localised in the sphere of voluntary movements, and do not effect the spheres of sensation, instinct, or intelligence. Innumerable observations on animals deprived of the cerebellum have shown the integrity of the special senses, while cutaneous and the muscular sense are similarily unaffected. Further, the two fundamental instincts — self-preservation and reproduction — are unaffected by this operation. The bitches on which extirpation had been performed became pregnant, passed through the periods of labour and the puerperium with such frequency that the laboratory seemed to be transformed into a maternity hospital! The author does not believe that there is any proof of depressed psychical functions after cerebellar extirpation. In apes the natural timidity appeared increased; and in dogs he often observed during the later periods a conspicuous indolence and listlessness.

But let us now analyse the *positive effects of cerebellar deficiency*, *i.e.*, the disordered voluntary movements which characterise animals deprived of the cerebellum. Of these there are two kinds, differing in nature and origin. They are (*a*) the true phenomena of functional deficiency, dependent on defective or absent cerebellar innervation; and (*b*) the phenomena of functional compensation represented by acts and movements,

instinctive or voluntary, by which the animal endeavours to repair the effects of the deficiency. Functional deficiency manifests itself essentially in the faulty *energy* which such animals employ in their voluntary movements, in the defective *tone* of the muscles and in the abnormal *manner* of accomplishing their contraction, while the compensatory acts are shown in the unusual *form* of the voluntary movements. Such is a brief description of the phenomena of cerebellar ataxy. Of this two typical and extreme forms can be distinguished: (1) that which follows extirpation of one half of the cerebellum, and (2) that which follows complete bilateral extirpation.

In the first division the features are of a hemiplegic nature, and are noticed on the same side as the lesion. The animal is unable to raise itself to walk, being only able to drag itself along on all fours, crawling on the buttock of the wounded side, and making the chief efforts with the limbs of the sound side. But on the contrary, during this period, the animal shows perfect capacity for floating, maintaining a good position, and of swimming with perfect co-ordination. It is constrained however, to turn towards the sound side, because it gives the water stronger strokes with the limbs of the sound side than with those on the side of the extirpation.

The dynamometric facts confirm the persistent deficiency of energy of the limbs on the side of the operation.

All the facts demonstrate that animals deprived of the middle lobe of the cerebellum, re-acquire after a certain time the capacity for walking without losing their equilibrium, this not depending on organic compensation, but upon the perfecting of certain instinctive or voluntary acts promoted by the sensori-motor sphere of the cerebrum, and determining functional compensation.

Certain of the phenomena of cerebellar deficiency which have been described have had the term *asthenia* applied to them. Conformably with extirpation of one half of the cerebellum, there is asthenia of the muscles on the corresponding side of the body, showing that the defective cerebellar innervation determines a *neuro-muscular hemiasthenia*. But this is not all, for associated with it is a depression of the normal tone of the muscles—*a muscular atonia*, and the following facts are illustrative of it. (*a*) Tremor in the muscles on the side corresponding to the extirpation, especially in the haunches during exertion. During rest or inactivity one notices greater flaccidity and less tension between the sound and wounded sides. (*b*) When the animal is eating, its limbs being abducted so as to enlarge its

base of support, and with its attention rivetted on its food, it has often been observed that the limbs on the defective side little by little gently flex; this produces a loss of equilibrium, and the animal falls towards the wounded side. (*c*) When walking there is a tendency to fall towards the defective side, which is due, not so much to inconsistent placing of the limbs of the wounded side, as to the sudden relaxation of the muscles of this side which the animal does not know how to obviate by compensatory actions. (*d*) Isolated elevation of the limbs of the wounded side as if the animal were ascending a small staircase.

Besides the asthenia and atonia, still another set of phenomena come under the heading of cerebellar deficiency. Such are the *tremors*, which are observed after the irritative effects have passed off. When the animal is walking quietly, they are most marked on the side of the extirpation, especially in the muscles of the limbs and of the vertebral column. They are evidence of a characteristic want of fusion and continuity of action of the muscles of the limbs and of a want of stability and firmness of the vertebral column. This also determines a certain amount of shaking, to which clinicians have applied the term, *titubation*. Under certain circumstances, for instance, when the animal is in the erect posture, or when greedily taking food, these tremors are exaggerated and assume the character of *rythmic oscillations*. The tremors, titubation, and rythmic oscillations are classified under the term *astasia*, and if limited to one side they form the condition known as *hemi-astasia*.

The phenomena of deficiency following bi-lateral extirpation are diffused equally over the muscles of both sides; but there is noticed as a result of this operation a peculiar form of gait known as the drunken gait ("démarche de l'ivresse" of the French authors).

The phenomena of degeneration following cerebellar extirpation will be discussed later on.

The experiments show that the cerebellum is not an organ intercalated among the great paths of the cerebro-spinal system and as such transmitting voluntary impulses from the cerebrum to the spinal cord, but that it is an appendix, by the side of the cerebro-spinal paths, being essentially an auxilliary or reinforcing organ for the central nervous system, which is more developed, the higher one goes up the vertebrate scale. Complete destruction of this organ does not produce paralysis of any groups of muscles; a proof of this is found, when stimulation by electricity of the sensori-motor sphere of the cortex provokes the same reaction as if

the cerebellum were untouched. Of the greatest importance in this relation is the intimate anatomical connection which the cerebellum has with the grey matter of the pons Varolii by its middle peduncles. With this knowledge we cannot but think, that the traumatic and inflammatory irritation of the peduncular fibres is transmitted directly to the ganglionic elements of the pons.

There only now remains to briefly consider certain trophic changes. For the first few days after the operation there was noted in some of the animals polyuria, glycosuria, and acetonuria. But apart from these temporary conditions during the course of the ataxy, certain general and local trophic phenomena were observed. Marasmus was occasionally present without any evident cause for its onset. Alopecia, erythema, and eczema, were noted in several cases at various periods. Conjunctivitis, keratitis, and otitis, were observed on several occasions. It is interesting to note that these solutions of continuity readily healed under the use of disinfecting lotions. Fatty degeneration of the muscles of the extremities was also seen associated with increase in the number of the nuclei of the sarcolemma.

Several chapters are given over to detailing and criticising the various theories of the functions of the cerebellum which have been expounded since the time of Willis.

I will conclude this review by briefly referring to the last chapter of the work, which the author devotes to "the first lines of a new doctrine." The results of the researches show that the cerebellum is an organ of bilateral function, but with mainly a direct action, in this way differing from the cerebral hemispheres, which also exert a bilateral action, but mainly crossed. The experiments also indicate that cerebellar influence is not limited to the muscles which regulate the different forms of posture and locomotion, but extends to all the voluntary muscles. They further clearly show that the middle lobe is not of greater functional value than the lateral lobes, and that, as a rule, the different portions of the cerebellum have the same functions, and that the cerebellum is not a collection of centres functionally distinct or different, or that the various segments have an intimate or direct influence over special muscular groups, but that the influence which the cerebellum normally exercises over the system is a sthenic, tonic, and static, neuro-muscular action. It is also evident that the cerebellum transmits, by its efferent paths, a trophic action, direct and indirect. The direct is shown in the degenerations and scleroses which follow its ablation, the indirect by the muscular and cutaneous dystrophies which have been recorded. The functional influence, which the

cerebellum transmits normally to other nerve-centres, is slow, quiet, and continuous. But the experimental, as well as the clinical evidence, shows that in morbid states, both the trophic and functional activities are increased, so as to produce violent perturbation and disturbance of the sensorial, motor, and trophic functions. The sensorial disturbance consists essentially of vertigo, the motor of those multiform aberrations which produce motor inco-ordination, while the trophic finds expression in polyuria, glycosuria, acetonuria, and rapid decrease in weight. The trophic and functional influences probably represent different aspects of one complete physiological process, of the nature of which we are ignorant.

The functional action of the cerebellum is different from that of the other parts of the nervous system. The effects of cerebellar deficiency have been shown to be asthenic, atonic, and astatic, while those of other centres show complete or incomplete paralysis of motion and sensation. The reason of this is that this organ forms a small, relatively independent system, defect of which does not interrupt centripetal or centrifugal conduction between the cerebrum and the peripheral nervous system. It is to be regarded as a coadjutor or reinforcing organ to the great cerebro-spinal system.

The brains and spinal cords of the animals (dogs and monkeys) on which the preceding observations had been made, were handed over to Marchi, for the purpose of following out the degenerations which resulted from the extirpation of whole or part of the cerebellum. For this purpose he used Weigert's hæmatoxylon solution, and a method which is now known as Marchi's method. Briefly stated, this consists in hardening the tissue in Müller's fluid. After a short time pieces the size of 1 c.m. are cut, and placed for eight to ten days in a mixture of Müller's fluid (2 parts) and osmic acid (1 part). The tissue is then prepared for the celloidin process. The degenerated tissue is stained black.

1. The degenerations which result from extirpation of one-half of the cerebellum (*e.g.* right hemi-extirpation) are :—

(*a*) In the region of the superior peduncles the osmium-bichromate method showed evidence of degeneration in the right superior cerebellar peduncle, and partly also in the left; much degeneration of the left red nucleus, and slightly of the right, while in addition there was evident alteration in the fillet, many of the fibres of the third pair of nerves, the posterior longitudinal bundle, some of the fibres of the pyramidal tracts in the crura cerebri and degeneration of the right optic tract.

(*b*) In the region of the middle peduncles there was found complete sclerosis of this peduncle on the side of the extirpation, the degeneration passing over the middle line. In addition there was found degeneration of the nerve fibres of the fifth pair on the same side of the fillet, of the posterior longitudinal bundle, and of a small band of fibres which lie posteriorly and externally to the superior peduncles. Sclerosis of the grey matter of the pons (nuclei pontis) was also observed.

(*c*) In the region of the inferior cerebellar peduncles the following degenerations were brought out by the osmium-bichromate method: The internal portion of the inferior peduncle, the external portion of the restiform body, certain of the striæ acusticæ and of the external auditory nucleus, many fibres of the ascending root of the fifth nerve, the fillet, the inter-olivary layer, and the posterior longitudinal bundle. Some of the fibres of the hypoglossal nerve and of the pyramids were also degenerated. In sections treated by Weigert's method the degeneration is not so diffuse, but there is evidence of degenerative change in the olive of the opposite side, the corpus restiforme of the same side, the direct cerebellar tract, and the fillet.

(*d*) In the spinal cord the author detected degenerations by the osmium-bichromate method. The Weigert method was not so successful in bringing out this degeneration. The tract in question occupies the antero-lateral region of the cord, one portion being in connection with the anterior part of the direct cerebellar tract, the other in connection with the pyramidal fibres on the same side as the extirpated cerebellar hemisphere.

As a result of ablation of the middle lobe of the cerebellum the author found the degeneration in the superior peduncles scarce and limited; but there was much alteration found in the fillet, the posterior longitudinal bundle, and the roots of the third nerves and the fibres of the optic tracts. From this one gathers that the fibres of the superior cerebellar peduncles come mainly from the nucleus dentatus and the cortex of the lateral lobes. All the transverse fibres of the middle peduncles are degenerated. The degeneration in the inferior peduncles are limited to the lateral part of the corpus restiforme. There is also degeneration of the fibres of the corpus trapezoideum and of the roots of the third, fifth, eighth, and twelfth cranial nerves. In the spinal cord there was sclerosis of the antero-lateral columns.

The author summarises his results in the following conclusions:

1. The superior cerebellar peduncles do not completely decussate, a small band of fibres passing to the optic thalamus on the

same side, the principal mass terminating in the red nucleus of the opposite side.

2. The middle cerebellar peduncles are not merely commissural strands from one hemisphere to the other. Many of the fibres enter the pyramidal bundles and end in the grey matter of the same side, while others pass to the grey matter of the opposite side.

3. The inferior cerebellar peduncles send a tract of fibres to the opposite olivary body. They are in all likelihood formed of afferent and efferent fibres.

4. The posterior longitudinal bundles and the fillet arise from a common origin in the middle lobe of the cerebellum. They course with the middle peduncles and come into relation with the nuclei of the cranial nerves, the nuclei pontis, the corpora quadrigemina, and probably also the corpus striatum. At the level of the olive the posterior longitudinal bundles fuse with the fillet and thus form a connection with the antero-lateral regions and the anterior horns of the spinal cord.

5. That the cranial nerves are closely related to the cerebellum through the medium of the fillet and the posterior longitudinal bundles.

6. The origin of the three peduncles is diffused over the cerebellum, but particularly the nucleus dentatus furnishes most of the fibres of the superior, the middle lobe of the middle peduncles.

It is impossible as yet to criticise the results which Marchi has obtained and which are briefly recorded above. The osmium-bichromate process of staining, which he has adopted, and which is now known as "Marchi's method," is, as far as my experience goes, open to numerous fallacies. The manifold connections of the cerebellum which he has described will, therefore, require ample corroboration before they can be finally accepted.

WILLIAM ALDREN TURNER.

Principles of Psychology. By W. JAMES. 2 vols., 8vo, pp. xii., 689 and 704. London: Macmillan and Co., 1890.
Text-Book of Psychology. By W. JAMES. 1 vol., 8vo, pp. 478. London: Macmillan and Co., 1892.

Prof. James' abridgment of his "Principles" is not so much a text book of psychology as a text-book of the author's psychology. So much concession he has made to criticism and general usage

that the treatment of Sensation comes first in the book. He has also taken into account the physiological ignorance of the average student of psychology, and furnished a chapter on the structure of the brain, and descriptions of the various sense-organs. The rest of the work follows, with omissions, the plan of the "Principles."

The author's working hypothesis is that "mental action may be uniformly and absolutely a function of brain-action, varying as the latter varies, and being to the brain function as effect to cause." Not that he is a disciple of the "new psychology." The question of free-will is to be ignored in psychology, being psychologically unanswerable (*Text-Book*, pp. 456, 458); but ethically the will is free (p. 461). "At present psychology is on the materialistic tack, and ought in the interests of ultimate success to be allowed full headway, even by those who are certain she will never fetch the port without putting down the helm once more" (p. 7). Psychology as a natural science is "a string of raw facts; a little gossip and wrangle about opinions; a little classification and generalisation on the mere descriptive level; a strong prejudice that we *have* states of mind, and that our brain conditions them: but not a single law in the sense in which physics shows us laws, not a single proposition from which any consequence can causally be deduced" (p. 468). Thus introduced to his science, the beginner in psychology will surely not catch the enthusiasm for it which the book of a thorough believer in the new experimental direction would give him. Prof. James himself possesses this enthusiasm in so eminent a degree that during the reading of him the paedagogic defect will not appear. But afterwards, when the factual content of the book comes to be sorted out from its setting, and the student has forgotten the phrases and images that are so telling, he will, one thinks, be either prematurely cynical or "a little mixed."

At the same time, the author is true to his plan, and the whole exposition has a neurological savour. His general attitude towards psychology differs from that usually taken up by the empirical psychologist. Instead of building up consciousness out of its units, Prof. James prefers to take the entire conscious state, as it is given, and to work at it so in the concrete. It is doubtless good that the student should be energetically reminded of the abstract nature of the "sensations" and "feelings" and "impulses" with which most of the modern books begin. But the author's method leads him at times to vagueness; to the substitution of suggestive analogy for laborious analysis; and to a fine impatience with (here and there combined with misunder-

standing of) exact experimentation. And this frame of mind, again, is not the best for the beginner.

So much by way of introduction. The chapter on "Sensation in general" bears marks of haste. On p. 14 we read: "The first time we see *light*, . . . we *are* it rather than see it." On p. 15: "The very first sensation which an infant gets *is* for him the outer universe." Which standpoint, one may ask from the beginner's point of view, is to be adopted, or are both? A similar inconsistency occurs on pp. 10, 11, with regard to the law of specific energies. Attention may be called here to the repetition on pp. 73, 413, which was probably not purposed. As to Fechner's law, Prof. James is more than sceptical. "The many pounds which form the just perceptible addition to a hundred weight feel bigger when added than the few ounces which form the just perceptible addition to a pound" (p. 21). But this is just the point: *do* they? One must hope that the student will not rest satisfied with a dictum, but will go into the matter for himself. The author's thesis that "each sensation is a complete integer" is not interfered with by Fechner's formula, as he thinks it is.

There follow chapters on sight, hearing, touch (with the temperature sense, the muscle sense, and pain), and sensation of motion. A large portion of these is adapted from Martin's "Human Body." Taste, smell and the common sensations are omitted "as almost nothing of psychological interest is known concerning them"! The whole account is rather introductory than systematic; hence such confusions as the treatment of pain under the head of sensation (elsewhere it is a primitive emotional species; p. 183), or the description of tone-pitch as a "quality of feeling." When the author comes to write his promised chapter on pleasure and pain, he will probably give a different psychological explanation of the facts of analgesia.

The sketch of the structure of the brain is clear and good; but a word might have been said as to the functions of the parts described. As it is, the chapter on brain function stands in rather loose connexion with that on structure, the cerebral cortex alone being treated at any length in the former. This is, of course, conditioned in part by the necessity of condensation; the main point for the writer being the psychological implications of brain development. And, regarded by itself, there is nothing exceptionable in the plan of the function-chapter; unless indeed it be the instance of the animal whose "life again and again pays the forfeit of his gluttony."

Under the heading "general conditions of neural activity" are brought together the subjects of nervous discharge, reaction-time, summation of stimuli, cerebral circulation and cerebral thermometry. Martius' results have not convinced Prof. James that the "muscular" reaction is not a reflex. But if this is so, how can it have a *comparative* value, for the determination of choice times, discrimination times, &c.? What we get by simple subtraction of the "sensorial" time may be doubtful (as regards certain constituents); but what we get by subtraction of the "muscular" time is a mere number, the difference of two psychological incommensurables.

There follows a chapter on habit, which is treated from a physical point of view. The discussion is exceedingly fresh and stimulating, though it is as much paedagogical and ethical as psychological, and presupposes an ethical theory. The reader will be reminded of Hering's physical treatment of memory.

It has already been said that the author advocates an analytical treatment of conscious processes, in opposition to the synthetic arrangement usual in text books. The fundamental fact for him is not the sensation or the impulse; it is "that thought goes on." Every thought is owned. No thought once gone can recur; it is the object that is got twice. ("Helmholtz calculates that the white marble painted in a picture representing an architectural view by moonlight is, when seen by daylight, from ten to twenty thousand times brighter than the real moonlit marble would be. Such a difference as this could never have been sensibly learned." The writer does not seem to see how directly this tells against his criticism of Fechner). Each personal consciousness is sensibly continuous, is a "stream of thought." Where Spencer writes that mind is composed of feelings and relations between feelings, Prof. James would say that it is made up of substantive feelings and transitive feelings (feelings to which the relations between objects are known). Every image is surrounded by a halo of relations, a "fringe." Lastly, the thinking consciousness is selective.

Psychologically regarded, the discussion of "the self" is a characteristic piece of descriptive analysis; but it trespasses also upon theory of knowledge and, *pace* the author, upon metaphysics. Noteworthy, when one considers the rigorous aloofness of the German experimental school, is the expression of his conviction that "a serious study of trance-phenomena [mediumships, &c.] is one of the greatest needs of psychology." The physiological ground of the "narrowness of consciousness" is found in the fact

that "the activity of the hemispheres tends . . . . to be a consolidated and unified affair. . . . . determinable only as a whole;" that of attention is threefold,—ideational and sensorial excitation of the appropriate cortical centre, afflux of blood thereto, and adaptation of the sense-organ.

Conception, the function by which we discriminate and identify, is next dealt with. The author maintains the existence of a real sensation of difference. Association is originally treated. We are to talk of association, as effect, between things thought of; as cause, between brain-processes. The elementary causal law is the law of neural habit. Psychologically expressed, Prof. James' view of "association of ideas" would be a theory of association by contiguity. *What* contiguous ideas are associated to a present one depends, of course, on many moments. Simple similars do not associate; and compound similars are partially identical. The author suggests a new nomenclature; total, partial and focalised recall; the latter representing the "association by similarity" of the older empirical psychology.

The condition of our sense of time is awareness of change. When nothing else is before consciousness, it is the organic sensations and their feelings, or the pulses of imagination and attention, that constitute the changing process which is its object. Retention and recollection are explicable in terms of the same law of neural habit which was called upon for the explanation of association. A "slumbering" condition of an organised neural path means retention; its activity means recall. The brain-tracts excited by the event and by its recall are in part different. On the other hand, the tracts for imagination and sensation are probably the same.

Sensational and reproductive brain-processes in combination give us the content of our perceptions. The process in illusion and perception is the same; what is false in illusion being not what is given, but what is inferred. The causes of illusion are ingrained association or absorbing interest. Under these conditions suggestibility obtains in all the senses. The common view that in the case of hallucination there is no objective stimulus at all is rejected, though it is admitted that centrally initiated hallucinations are theoretically possible. Physiologically considered, Binet's theory seems, I think, the more probable; but it is very difficult to get conclusive evidence.

In the chapter on "Perception of Space" the author develops his theory that voluminousness (spaciousness) is a primary attribute of sensation. He has Dr. Ward on his side;

but the view is opposed to that of most experimental psychologists. This "extensity," which is not to be confused with the psycho-physical sensation-attribute of extension, is the original space-sensation (so that the title of the chapter is a misnomer); and out of it comes exact spatial knowledge, by way of discrimination, association and selection.

The chapter on Reasoning contains some suggestive remarks on animal psychology. In those following, Prof. James elaborates his thesis that "all consciousness is motor." Emotions and "instincts" are brought into close relation with one another. "The only distinction one may draw is that the reaction called emotional terminates in the subject's own body, whilst the reaction called instinctive is apt to go farther and enter into practical relations with the exciting object." As regards emotion, the at first sight paradoxical position is taken up, that the mental state results from the bodily expression. "The bodily changes follow directly the perception of the exciting fact, and our feeling of [*i.e.*, our conscious experience of] the same changes as they occur is the emotion." I paraphrase the word "feeling" as the sense seems to require; it is very loosely used in the discussion. The special nervous processes involved are sensational. The accidental factor in the genesis of the emotional reaction is strongly insisted on. Instincts are the "functional correlatives of structure." Every instinct is an impulse: is as closely connected with will, that is, as (through emotion) with feeling. Human instincts are so many that they block each other's paths; hence the characteristics of "intellectual" life, hesitation and choice. Very interesting is the author's interpretation of the two "principles of non-uniformity," the inhibition of instincts by habits and the transitoriness of instincts.

Since reflex, instinctive and emotional movements are primary, it follows that voluntary action is secondary. (There crops up at once in one's mind the opposed statement of Wundt's: "Reflexes are voluntary acts which have become mechanical.") The first condition of voluntary life is the presence of ideas of movement, memory-pictures of sensations of motion, or of sensations aroused in the special sense-organs by motion. These ideas are the "cues" of ideo-motor action, which is only another instance of the impulsive nature of consciousness. To them must be added, in certain circumstances, the "*fiat*, or element of consent or resolve," which comes in "when the neutralisation of the antagonistic and inhibitory idea is required." This *fiat*, the essential phenomenon of will, consists in the effort of attention,

and in the attitude of "consent to the reality of what is attended to." (*Principles* ii., 568.)

With the "Epilogue: Psychology and Philosophy," the introductory sentences above have already partially dealt. It contains paragraphs on the word "metaphysics," on the "relation of consciousness to the brain" and of "states of mind to their objects," and on the changing character and unverifiability of conscious states; and is sufficiently pessimistic. In one way it will be useful, as serving to remind the reader of the immense complexity of the whole subject under discussion, and so to correct the impression of simplicity apt to be left by schematic figures and explanations.

Implicitly, of course, a portion of the "Principles" has now been passed under review. Those who have read the above notice of the text-book must have felt a growing conviction that a thorough criticism of Prof. James' work must be psychological and not psychophysical or neurological, despite his natural-science standpoint and physiological theories. If this is true of the smaller book, it is much more so of the "Principles." Prof. Ladd goes, no doubt, too far, when he talks of the "exceedingly thin and dubious diagrammatic representations of brain-processes occasionally interjected into the discussion of psychological phenomena;"[1] but more than one recent work has shown how indefinite cerebral physiology still is, and I do not think that Prof. James has made the most even of the materials that are to hand. For a detailed psychological criticism this is not the place. I will only briefly refer to the chapters of the larger book, which are unrepresented in the abridgment.

In the first volume there are four such: those on the "automaton theory," the "mind-stuff theory" (partially repeated in the Epilogue to the text-book), the "methods and snares of psychology," and the "relations of minds to other things." The first-mentioned theory, according to which consciousness, at a certain stage of evolution, supervenes upon, but cannot interfere with, the sequence of autonomous neural processes, is rejected on the ground that conscious direction of the nervous system is useful. "But if it [consciousness] is useful, it must be so through its causal efficaciousness, and the automaton theory must succumb to the theory of common sense." The mind-dust theory is also rejected, on the score of the simpleness of mental states; any "integration" or "composition" which takes place is subconscious and physical. (The chapter contains a good discussion

[1] *Philosophical Review*, i. 32, 33.

of the "psychology of the unconscious.") There remains the multiple-monad hypothesis, which is physiologically more than improbable; and—the soul. The author inclines to doubt the existence of the latter; though for psychological purposes he regards it as safer to rest content with the bare fact of psycho-physical parallelism. Psychological method is that of introspection, experiment, and comparison; psychological errors arise from the misleading influence of language, and from the psychologist's "confusion of his standpoint with that of the mental fact" which he is regarding.

The last, rather oddly-named chapter, treats of the temporal and spatial relations of mind, and of its relations to other minds or to material things. In discussing the question: "are we ever wholly unconscious?" Prof. James adopts the somewhat crude terminology of the French writers, and speaks of the "primary" and "secondary self," of mind being "split up into separate consciousnesses," as if this were really intelligible language. The question itself he wisely leaves open.

Three chapters call for notice in the second volume: those on the "perception of reality," hypnotism, and "necessary truths and the effects of experience." Belief, like consent, is declared to be a psychosis *sui generis*. Indeed, the two are perhaps at bottom one; for "will and belief, meaning a certain relation between objects and the self are two names for one and the same *psychological* phenomenon." As experienced, belief is a "sort of feeling, more allied to the emotions than to anything else." The author digresses into theory of knowledge to discuss at length the nature of psychological "reality."

Of the three theories of the hypnotic state—animal magnetism, neurosis and suggestion—Prof. James prefers the last: "provided we grant the trance-state as its prerequisite." The chapter is not very satisfactory.

Finally, we have the chapter on psychogenesis, where Prof. James separates himself sharply from the experience-philosophy school. "The features of our organic mental structure cannot be explained at all by our conscious intercourse with the outer environment, but must rather be understood as congenital variations, 'accidental' in the first instance, but then transmitted as fixed features of the race." As regards our consciousness of classificatory, logical and mathematical relations between ideas, Spencer's views are declared "unintelligible." Darwin's explanation of the origin of instincts—by the "natural selection of accidentally produced tendencies to action"—is adopted. On

the question of the inheritance of acquired characteristics the author had arrived at an anti-Lamarckian position, independently of Professor Weismann. It is curious that the discussion moves always on the same lines, and that a theory such as that of Nägeli receives so little attention.

It is unnecessary in conclusion to express any opinion upon the general merits of the work. Prof. James' rank among the foremost of contemporary psychologists has been universally recognised. There is much in his volumes to criticise; and his attitude to the most modern and hopeful direction of psychological investigation is, unfortunately, not that of complete sympathy. But, all defects counted, there hardly exists a so stimulating and thought-compelling treatise in all the voluminous literature of the science.

The Human Mind: a Text-book of Psychology. By JAMES SULLY, M.A., LL.D. London: Longmans, Green & Co., 1892. 2 vols., 8vo, pp. xvii., 501, 393.

There can be no doubt that the recent ferment among psychologists—three important treatises, not to speak of new editions and minor works, have appeared in English within the last four years—has been conditioned to a large extent from without. The two new sciences of psychophysics and physiological psychology are the avowed *motiv* of Prof. Ladd's compilation (previously reviewed in BRAIN), and in the preface to the present volumes Mr. Sully makes them a determining factor in his recasting and expansion of the *Outlines*. Not that this is a sufficient description of the new text-book, which is, as the author claims, an independent work. Clearly and moderately written, and (except in one debateable regard) well proportioned, the book is a welcome addition to the literature of our empirical psychology, and will be indispensable to students of the science. If this statement seems weakened by the following criticism, it must be remembered that the psychologist and the psychophysicist cannot, in the present state of things, be expected always to think alike. There is only too much room in the sphere of mind for difference of opinion. Moreover, it is hardly possible for one man to know the literatures of three sciences equally well.

The first part of the work—"introductory"—consists of three chapters, which deal respectively with the "aim and scope" of psychology, its "data and method," and with the "physical basis of mental life." The author rightly insists upon the disparity of mental and physical processes, and upon the distinctness of psychological method. When he says, however, that "all that the scientific psychologist can mean by the phenomenal connexion of mental and bodily processes is a relation in time" (I. 47), he is, I think, ascribing to the psychologist a metaphysic which is directly opposed to his practice. Prof. Ladd, though he has gone far beyond his last, did good service to psychology in pleading for the recognition of causal interaction of "mind" and "body" as a working hypothesis in psychophysics (Elements, 648 ff). Mr. Sully cannot himself avoid dropping into terms of definite causal relation in the course of his exposition (see, *e.g.*, I. 29, 125, 135, 141, 284; II. 38, 275); and such expressions as "physiological ground," "physiological conditions" are at least ambiguous. The law of conservation of energy (II. 367-8) need not be violated by such a provisional assumption; for we may imagine the series of events to be AB—cd—EF, where cd is the psychical process, and yet none of the physical energy of AB is lost in EF. The logical and metaphysical difficulty of course remains untouched, but need not concern psychology.

It is difficult to say how much of the results of brain physiology should be incorporated in a psychological text-book. Mr. Sully has expressed his opinion on the subject elsewhere (*Mind*, xvi., 396). The psychologist should, on his view, "confine himself to a bare summary of the more important results of anatomical and physiological research, so far as these have a plain bearing on the understanding of physical processes, a bearing which he means afterwards to illustrate." It is no doubt unnecessary that a certain portion of physiology should be transcribed by every writer of a psychological text-book. But there is unfortunately in English no concise exposition of the physiology of the central nervous system, written by a competent hand expressly for students of psychology, such as prefaces Wundt's *Grundzüge*—now five years old. I cannot but think that such an exposition, brought up to date, would be infinitely more valuable than the main bulk of the schematic and metaphorical neurology which is to be found in these two volumes. At the same time, in the little which he has written in this introduction on the nervous system, Mr. Sully has discriminated wisely. The slender experimental basis of the theory of functional indifference of nervous

elements—probable as the theory in itself is—should perhaps have been noted. The distinction between motor and sensory cells on the ground of size can hardly hold absolutely. There are good reasons why the largest cells should be motor; but none why motor cells should not also be small. More important is the fact of the bipolarity of the cell, as established by Ramón y Cajal's researches. Lastly, the facts of localisation might have been with advantage given in a little more detail; or at least first-hand references furnished to some of the work done since 1886.

Part II. ("General View of Mind") contains four chapters, in the first of which mind is analysed into its elementary functions. The value of such analysis had been well emphasised in the introduction. Mr. Sully classifies mental functions as knowing, feeling, and willing. In all *knowing*, two stages are recognisable; the fusion of presentation with conation, and the stage of intellection proper. The latter again is three-sided, appearing as discrimination (relationing), assimilation and associative integration. A necessary condition of intellection in general is the mental property of retentiveness. The root-function in *feeling* is sensibility to pleasure and pain. The two directions of *willing* are attention and voluntary movement. All three processes are concerned in every psychosis.

This tripartite division is, as the author admits, "the outcome of subjective analysis, unaided by objective (physiological) considerations." Indeed, as regards the special function involved in intellection, Mr. Sully parts company with psychophysics. The psychophysicist, though allowing that such an analysis as the above is descriptively correct, cannot go beyond his elements; but must derive the complicated mental processes from the interaction of sensation, feeling and conation, aided by reproduction. Lewes' analysis of mental functions may stand, if for "sensible affection" we write "sensation and affection." Herbert Spencer, in regarding mind as composed of feelings and relations between feelings, saves his biological consistency by the vagueness of the word "relation," which varies between the German *Verbindung* and *Beziehung*. Mr. Sully's language tends also to be ambiguous at times; as when he substitutes the possibly passive "differentiation" for "discrimination," uses the parallels of organic segmentation and co-ordination, or talks of mental elaboration as "mechanical." In one passage the essence of the discriminative process seems to be placed in its conative side (I. 174; cf. 170). But that knowing involves a peculiar functional activity of mind which has no physiological analogue, is directly admitted for dis-

crimination and assimilation (*e.g.*, I. 171, 193, 203): though these statements again are more or less invalidated by a later one (II. 367).

The ultimate psychical elements are sensations, sense-feelings, and certain active tendencies. Mr. Sully, for the most part, sharply distinguishes between sensation and feeling, though at times he falls a victim to the looseness of the English terminology. In the note on Weber's law, Wundt's position is too closely correlated with Fechner's; it is purely psychological. A reference to Müller's *Grundlegung* for the physiological view might have been given here. The author has overlooked Aronsohn's researches on smell. Hering's theory of thermal sensations deserved a little more attention. It is surely inconsistent to make timbre a quality of *tone* sensation, while explaining that it only attaches to clangs (I. 91, 112). On pp. 112, 114, the conclusions of Prof. Stumpf's "investigations" are curiously misrepresented (cf. *Tonpsychologie*, II. 56, 525, 537). The connection of appreciation of direction with affection of the semicircular canals must be given up, if for no other reason, on mechanical grounds. The interaction of chromatic and achromatic qualities in colour-sensation requires more exact investigation before one is able to write so certainly as Mr. Sully does. It is not clear whether he recognises quality-differences in the *Helligkeitsreihe*. That sensory fibres are present in muscle (though not to the extent to which they occur in the tendons) may be taken as proved; though their signification for sensation is by no means clear. The author rightly rejects the hypothesis of pain-nerves (II. 14), but without referring to the best discussion of the question from the psychological side (Külpe's *Zur Theorie der sinnlichen Gefühle*). He inclines towards a belief in sensations of innervation. Here again the references are insufficient. Indeed, one may take this opportunity of saying that the space-saving method of quoting text-books, where possible, has been too exclusively followed in this part of the book. In the case of Prof. Ladd's transcriptions it becomes positively irritating. Would it not be better to refer the student directly to one or two classical researches in the various departments, from which he could find his way to the whole literature? On the other hand, Mendoza's book might have been cited, in place of Bleuler and Lehmann, for coloured hearing. The printing of "experience of movement," and "experience of impeded movement" in the text appears to imply a theory which is contradicted by the page-headings,

"sensations of movement," "sensations of resistance." Both views have found representatives in psychophysics. All these are, of course, minor points, and do not affect the value of the exposition as a whole.

The two following chapters deal with mental elaboration; and the first with attention. Attention in its elementary stage is described as a reflex reaction of conation upon sensation, which is accompanied and coloured by strain-sensations and sense-feelings. This is, pretty exactly, Wundt's passive apperception. That the process should be thus early emphasised and discussed is a great help to the student; and Mr. Sully's treatment is full and clear. I would only take exception to the view, that the concomitant muscular sensations can be made the measure of sensational attention; and suggest the selection of other reaction-times than those adduced on p. 155. As these stand, they are calculated to give the reader a totally false idea of the absolute duration of the simple reaction. All physiological theories of attention are as yet necessarily unsatisfactory.

In the account of differentiation (discrimination) and assimilation, Mr. Sully's language, as has been pointed out above, is not quite clear. He is, perhaps, hardly just—whether from the psychological or physiological standpoint—to those who refer our apprehension of likeness to partial identity. The psychical spring from like to like is, I think, the more "speculative hypothesis" of the two; and there is a great deal to be said in answer to Stumpf. The author recognises but one law of association of ideas: that of contiguity (I. 294 ff). There seems to be good reason for hoping that this view will soon win general acceptance among psychologists. When we have once got so far it is, more or less, indifferent whether we, for the present, describe the other constituent in the reproductive process as "assimilative revival," (*Widerherstellung des Gleichen*), or as a case of substitution conditioned by interest, &c. A long series of experimental researches needs to be carried out, before the question as to its nature can be decided. It seems somewhat premature to regard fusion (*Verschmelzung*) as an instance of close association. The physiological basis of reproduction is, as Mr. Sully says, quite conjectural.

I have devoted so much space to the first two hundred pages of the book, because there is here given in outline the system which is elaborated later. The three divisions of the work which follow—"Intellection," "the Feelings," and "Conation or Volition"—present in detail the doctrine here set forth in the rough. Mr.

Sully has had it in mind throughout that he is writing a text-book. His conclusions are cautious, and will no doubt be regarded as eclectic by extremists of all psychological schools. It is, however, most important that the student should not be led "to take sides" from the outset; and the knowledge that there are two views on most psychological questions cannot be too deeply impressed upon him. The most serious blemish of the book is its hypothetical physiology. In his desire to translate psychical processes into terms of neurology, the author makes the latter vague, and even metaphorical. The reader is struck rather by the simplicity of the supposed cerebral action than (as should be the case) by its enormous complexity. Moreover, the translation has as yet, in most instances, very little scientific justification.

The section on "Intellection" begins with a chapter on perception. In quoting Helmholtz on p. 213, Mr. Sully seems to have followed Prof. James ("Principles," I. 169, II. 111) without referring to the original. The passage quoted from the *Thatsachen in d. Wahrnehmung* (1878) does not say what he makes it say.

On tactual space-consciousness he takes up a sound and moderate position, rejecting the primitive-bigness hypothesis as a psychological *ὕστερον πρότερον*. Wundt's theory of psychic synthesis is stated misleadingly as regards its intensive factor (*Grundzüge*, I. 406, II. 32. Mr. Sully repeats the mistake elsewhere; *e.g.*, II. 212). The thirty pages on visual perception are necessarily insufficient for complete exposition. The psychogenetic child of p. 246 is open to objection. The eyelid pressure of Münsterberg and Schwarz should perhaps be supplemented by the friction of the eyeball in its socket. The time-sense can hardly any longer be brought, without reasons given, under the heading of auditory perception, especially if Münsterberg's views are accepted. The last sentence on p. 272 should be lengthened by the words "or of a common ground-tone." In the fifth, *e.g.*, this element of relationship is important.

The following chapter on "reproductive imagination: memory" deals with the "image" (and the transition to it from the percept), "association of ideas," "representation of time," and "other forms of suggestion." The exposition is elaborate, occupying nearly a hundred pages. Here, where we are on such debateable ground, the value of the work as a text-book is especially evident. The chapter on "productive imagination" has evidently been a labour of especial love. The two final chapters of the first volume deal with processes of thought: conception, judgment and reasoning (knowledge). The preliminary account of attention renders the

discussion intelligible at this stage. Many hints are given towards a psychophysical theory (*e.g.*, of causal judgment and belief). The sharp distinction drawn throughout between logic and psychology is helpful: and the aid furnished by language to the child's attainment of an idea of self is well emphasised; it is a point too often overlooked.

The feelings are rightly regarded by the author as directly conditioned by stimulus. Whether feeling can occur independently of sensation, or sensation independently of feeling, is left undecided; though the answer would probably be negative (cf. II. 7, 11 with I. 77. The two thresholds are assumed to be coincident in II. 21: hence the criticism of Wundt). The physiological conditions of feeling are a state of general psychophysical relaxation, and diffuse central excitation following on stimulation of a sensory nerve. Very little is said with this: and one wonders that Meynert's theory is passed over. The existence of *gemischte Gefühle* is, I think, very problematical, as is that of emotive discharges which are wholly instinctive. The chapters on the emotions (and, in particular, the discussion of the æsthetic sentiment) are most interesting reading.

The last section of the work treats of conation or volition in three chapters, which deal with "voluntary movement," "complex action," and "concrete mental development." Primitive movements are classified as random, sensori-motor (conscious reflexes) and instinctive. It seems to be a point of honour with psychologists to have a special terminology and set of definitions in the sphere of volition. Mr. Sully tells us clearly what he means by instinct and reflex; and his remarks on impulse (*Trieb*) are especially good. His general position is that "the child finds his way to a large part of his voluntary pursuits by what we must call accidental coincidences." That is to say, given instinctive movement and sensational attention, voluntary movement will necessarily arise sooner or later under the influence of feeling. It must be noted here that "voluntary movement" has a very definite sense; volition at a certain stage is, of course, postulated with the word "attention," and the instinctive movements have themselves (as Wundt points out) the character of "simple impulse-movements." The author inclines to suppose that innervation of the antagonists plays a large part in the inhibition of voluntary action; but he does not mention Orschansky in connexion with this thesis. It is gratuitously inexact to speak of feeling at this stage of the discussion as being "concomitant *or effect*" of sensation and idea (II. 270). How absolutely conjectural the

physiological basis of volitional effort is may be seen by comparing Mr. Sully's view with that of Prof. Müller.

The book concludes with a series of fourteen short appendices mainly upon borderland questions, written from the historical point of view. The text is singularly free from printer's errors: but many words and a large proportion of the references in the notes require correction.

Handbook of Psychology. By J. M. BALDWIN. I., Senses and Intellect, 2nd Ed., 1890; II., Feeling and Will, 1891. New York: Henry Holt & Co. Pp. xv., 343; xi., 394.

That Prof. Baldwin's Handbook is a valuable contribution to Psychology, viewed from the educational standpoint, is shown both by the many favourable notices of it which have already appeared, and by the call for a second edition of the first volume within a year from its publication. And yet it is in this work, far more than in those previously spoken of, that the difficulty of assimilating the new manner of thinking to the old impresses itself upon the reader. One seems at times, in passing from chapter to chapter—less often within the limits of a single chapter—to spring from speculative psychology into psychophysics, without any aid from stepping-stones, and without any indication from the author that something in the way of a bridge is necessary. Prof. Baldwin will base his psychology upon "empirical investigation": "psychometry" [why cannot we get rid of this word?], "psychophysics and neurology" shall furnish the data for inductively established hypothesis. Then why is the account of the nervous system prefixed to the second volume; to the consideration of feeling, rather than to that of sensation? As regards the physiology of feeling, in the general sense of the word (I., 108; the author is very vague about it), we know next to nothing; of the physiology of sensation we know in comparison a great deal. I will not multiply instances. The grafting process is exceedingly difficult; the more so since there are signs that psychophysics means to expand into an experimental psychology, with new concepts as well as independent methods; so that the would-be conciliator to-day is almost forced into eclecticism.

The account of the nervous system, and of its connection with consciousness, occupies the first ninety pages of the second

volume. It represents a consistent attempt at a "philosophical" treatment; physiological and anatomical details are kept in the background,[1] laws and principles of structure and action emphasised. The fundamental specific properties of nervous tissue are neurility and sentience. Of neurility there are two current views: either it is the "molecular state which constitutes a course a good nervous conductor" (electrical analogy theory); or "the nervous system is a living organism, instinct with nervous force or neural properties throughout" (dynamic theory). Whichever view we hold, the central process is the important thing; for on the latter theory the activity of every element is "central." So we come to sentience, where we must distinguish the facts of integration (law of growth of living tissue), retention and selection. Of selection again there are two views: (1) "Does the nervous system select from a multitude of similar touches? The magnet selects from a multitude of similar filings, and the explanation seems to be the same. Neither the touches nor the filings are similar after all." (2) Sentience involves consciousness, and the *differentia* of the latter is choice. Prof. Baldwin accents as strongly as Prof. James the psychological significance of the law of nervous dynamogenesis—that every stimulus has a motor force.

Nervous reaction is (1) automatic (breathing, reproduction); (2) reflex: simple (winking) and co-ordinating (walking); and (3) voluntary. Negative nervous reaction covers the phenomena of inhibition. A curious error has crept into their discussion, in that the word *erschöpft* (exhausted) has been taken by the author to mean "formed, consolidated." There are five principles of nervous action. First stands that of specialisation of function. The questions arising out of the facts of cerebral localization, are shortly treated of, partly here, partly in the first volume. Their handling is not very satisfactory. Then follow the principles of functional indifference, substitution and specific connexion. This last means two things: that the presence of definite central or [and?] peripheral connexion alone determines the specific energy of a nerve-course, and that the connexions work by way of necessitating reaction to a specific stimulus.

[1] The statement as to the relations of nerve to cell, p. 6, must be altered in the light of more recent research. Cf., *e.g.*, Kölliker, Biol. Centralblatt, 30 Jan., 1892. I cannot but think, also, that a full-page diagram of conduction-paths (Flechsig's, *e.g.*), would, with advantage, take the place of Obersteiner's schema, p. 12, and of some of the text. Lastly, is it so certain that memory-pictures form part of conscious content in the case of the brainless carp (p. 27)?

"The only specific things after all are the stimulus and the movement." The principle of summation of stimuli seems to be regarded by Prof. Baldwin as furnishing the explanation of *Einstellung;* but it is hardly adequate to this. Finally, we have the three great laws of habit (consolidation, downward growth: diffused attention), accommodation (specialisation, upward growth: interest) and inheritance (the conjoint result of the two, acting in the race).

I have analysed this chapter thus at length, because of its strictly psychophysical turn. Whether the author's revival of Lewes' terminology will find general acceptance is, I think, doubtful. It seems unnecessary to go outside the usual physiological nomenclature, and a new source of confusion is introduced by using "sensibility" as equivalent to Lewes' "sentience," and *vice versa*. In the summing up, also, an unfortunate choice of terms has been made. Habit is, anatomically considered, "the arrangement of elements more suitably for a function, in consequence of former modifications of arrangement through that function." But what is this but one kind of specialisation—which Prof. Baldwin will make the very opposite of habit?

Is the "nerve-process, irritability," conscious; or is a certain degree of development necessary for the arising of consciousness? The former view, that of Lewes, Bastian, and (in a modified form) Wundt, is preferred by the author. He finds, rather boldly, a confirmation of it in the multiple personalities of hypnotic subjects: "the higher centres being here inhibited, . . . the lower supply experience which was before outside the conscious area." But here he seems going beyond his own definition of consciousness; and this is still more clearly shown in the following paragraph, where he writes, that probably "consciousness arises from the breaking down or expenditure of the cellular structure *in the highest centres*." So that the acceptance of Lewes' theory halts somewhat.

Consciousness is of three kinds. Passive consciousness is "subconscious sensibility;" simple awareness, unlocalised, effortless, purely affective. Yet sensibility itself is "sentience [regarded] as a conscious phenomenon" (II. 55); while elsewhere, the subconscious area is marked off from that of passive consciousness (I. 68). In reactive consciousness we are receptive, involuntarily attentive. Voluntary consciousness is deliberative, involves effort. The three degrees of consciousness are paralleled with the three phases of nervous function; but the physiological soundness of the division seems questionable.

Further special analogies are those of nervous retention and memory; of specific connexion and association; of summation of stimuli and, *e.g.*, Weber's law; of inhibition and the Herbartian idea-conflicts, or the mental processes consequent on suggestion. There follows a criticism of Dr. Maudsley's theory of the unity of consciousness, which is substantially a reproduction of an article published in *Mind*, in 1889, and some short remarks on mental heredity.

Another instance in this volume of the author's method is his discussion of theories of the sense-feelings, pleasure and pain. There are three groups of such theories; the static or absolute, the dynamic or relative, and the genetic. Physiologically, the former finds its expression in Mr. Grant Allen's definition of pleasure as "the subjective concomitant of the normal amount of function in any sentient tissue." Intellectually, it relates feeling to knowledge; and it is here that Prof. Baldwin parts company with it. In ruling out the Herbartian view, that "makes feeling dependent on the intellectual function considered as a play of representative elements or forces," on the ground that a sharp distinction of sensation from feeling is inadmissible, he is at variance with current psychological thought, which insists on just this distinction as primary. The second view, that of Ferrier and Dumont, says that all expenditure is painful, and all integration pleasurable. This the author meets by reference to the pleasure of novelty, which means pleasure in increasing expenditure. The genetic view is practically that of Aristotle. According to it, pleasure is the "conscious effect of that which makes for the continuance of the bodily life or its advancement." Is there not here again a mixture of psychological description, based on introspection and reflection, with psychophysical explanation? And does not Prof. Baldwin fall into the same error, as regards feeling, which "even the biologist Schneider" avoided, as regards consciousness in general?

In the remainder of the volume, which deals with the ideal feelings, and with the phenomena of will, there is much acute thinking, and a good deal that is original. The "Senses and Intellect" follows a more beaten track. After the introduction come two chapters on "general characteristics of mind," the second of which deals with Attention. It is a great gain to the student here, as in Mr. Sully's work, to have the latter process treated, at least in outline, thus early. The second part of the volume deals with the apperceptive function (presentation, representation, combination, and elaboration) and the rational function.

In the treatment of sensation, extension (*Ausdehnung*) is not included in the list of attributes (I. 85; but. cf. 109). The author decides in favour of the physiological interpretation of Weber's law, but, strangely enough, does not mention G. E. Müller in connexion with it. The account of chronometrical results is hardly adequate. One has no wish to carp upon this matter; but it is surely misleading to give .125″ as simple reaction-time, without naming the sense for which it holds, or stating that it represents the "muscular" form. The number itself seems to point to hearing-experiments. The account of the "theory of unity of composition of mind" would be better placed after the discussion of the organic theory of mental unity in the second volume.

Prof. Baldwin has taken a wide view of his science. He includes not only the two psychologies proper, empirical and rationalistic, but deals, in connexion with them, with logic, theory of knowledge and metaphysics. With those who hold that there is but one psychology, the empirical and experimental, he would agree to differ; they do not constitute the tribunal whose decision upon his work he would accept. A detailed criticism of his theories of sensation, &c., is therefore unnecessary. More serious are the objections which affect arrangement and terminology, and which have been already urged by other reviewers. Both faults can be corrected in future editions, and the Handbook will thereby be rendered even more valuable to the general student of psychology than it now is. An especially useful feature of the work is the list of "further problems for study," appended to each chapter.

An Introduction to Human Physiology. By A. D. Waller, M.D. London: Longmans, Green & Co., 1891. Pp. x., 612.

About half of Dr. Waller's book—eight chapters—is taken up with a consideration of the phenomena of excitation in the human body; and it is with this portion of his work that we are here concerned.

The author begins his exposition with a chapter on the general plan of the nervous system, in which he shortly illustrates the progressive elaboration of the neuro-muscular apparatus from the amœba to man. The nerve-centres in man are classified as

cortical and medullary or spinal. With the former are correlated voluntary actions, with the latter automatic and reflex actions. This correlation is, indeed, admittedly imperfect and, in special instances, arbitrary; for the physiologist is in no better case than the psychologist as regards a strict definition of the terms "reflex" and "automatic." In describing an automatic action as "a series of immediate motor reactions," Dr. Waller includes under it on the one hand such acts as breathing (primary automatic action) and on the other such acquired motor co-ordinations as are called into play in writing (secondary automatic actions). Such acts as walking occupy an intermediate place between the two main groups. In classing all these functions together the author varies from ordinary psychological usage, but is enabled to avoid the doubtful word "instinctive"

At the conclusion of the chapter is placed a paragraph upon the limitations of scientific enquiry. "The question *why?* is not answered by positive science, but only the question *how?* and sometimes the question *how much"?* It is quite true that no science can explain its first principles; and quite true that in the particular case before us the transition from "physical" to "psychical"—*e.g.*, from æther-wave to sensation—is wholly inexplicable. But one must not go too far. A proximate *why* is given by science in every case where she gives a more general *how*. The proximate *why* of human embryology is furnished biogenetically. In this sense the physiologist *can* "say why a muscle contracts," if he brings muscular contraction under the laws of pyro-electricity, *e.g.*:—by the way, there is no mention of Prof. Müller's theory in the chapter on muscle:—though for the physicist it is still only a matter of *how*.

The two following chapters deal with the peripheral nervous system, under the aspects of muscle and nerve respectively. "Muscles may indeed properly be regarded as the end-organs of motor nerves." This grouping is convenient; and though, perhaps, at first sight likely to mislead the beginner in physiology, will not cause any permanent misunderstanding, after the contents of the chapters have been digested.

After a useful description of electro-physiological instruments, and a short account of the histology and chemistry of muscle, the author proceeds to deal with its physical and physiological properties. For the study of the so-called muscular sense it is of the utmost importance to have in this regard an adequate and definite terminology. To judge from the footnote on p. 324 this seems to be as little the case in English as it unfortunately is in German. According to Dr. Waller, muscle is, physically re-

garded, extensible (*i.e.*, can be elongated by weighting, &c.) elastic (*i.e.*, shortens after removal of the weight), and viscous (*i.e.*, does not shorten to its unweighted length immediately, on withdrawal of the weight: the direct elastic shortening is followed by a gradual indefinite shortening). Physiologically considered, muscle is excitable; and this excitability manifests itself as contraction. It would, I think, be better here to use the word "excitation" and not "contraction" as the general term. A muscle may be excited in circumstances where contraction is impossible. But the main thing is to call the student's attention to the mutual independence of the conditions of muscular tension and excitation: and this the author does. In the rest of the chapter the main features of muscle-physiology are treated of. There is a good paragraph on the mechanics of muscular arrangement.

Dr. Waller is justly sceptical as to the existence of "pathic" nerves. He would, perhaps, have spoken still more decidedly, had he looked at the question from the psychophysical side, and realised what the hypothesis must mean, when every separate quality of the tone or colour scale can, and does, if carried beyond a certain intensity, pass over into pain. His remarks on "trophic" nerves are a model of careful reasoning. On p. 351, in connexion with the law of specific energies, a reference forward to p. 375 (where the functional indifference of the conductor is emphasised) would perhaps be in place. To the experimental proofs adduced for the latter might be added Babuchin's experiment on the electric nerve of *Malapterurus:* and du Bois-Reymond's name should have been mentioned. The exposition of the phenomena of electrical nervous stimulation is admirably clear; indeed, the author's account is by far the best of its compass with which I am acquainted in English or German.

A brief chapter on animal electricity gives a useful *résumé* of facts and theories from Galvani and Volta to du Bois-Reymond and Hermann.

There follows a more detailed review of the special senses. Curiously enough, Dr. Waller chooses to deal with colour entirely under the heading of the "physical data" of vision, though aware of his inaccuracy (p. 401). What he writes, however, is so much to the point that one can almost overlook this. His remarks on saturation and intensity of colour are not quite clear. He appears to maintain (what is not improbable) that colour-saturation depends on the amount of white light with which the colour is mixed: colour-intensity on the intensity of this white light. If this is not the sense of the passage, there is a con-

fusion between the physical and the psychophysical (or physiological!) aspects: if it is, it should be definitely pointed out that colour-intensity may be regarded as a function of saturation, and that the colour-scale presents sensational differences which are qualitative only. This is, indeed, plainly enough hinted by the diagram on p. 404; where a red is shown to be "dilute" or "dark" according as it is mixed with different quantities and qualities of the brightness scale.

Wundt's theory of colour-vision should have been mentioned on p. 407, if only to illustrate the all too suggestive fact that the progress of such theories has set steadily from the concrete to the abstract. A word might have been said, also, on the errors of perimetric experimentation. Moreover, as the sentence on p. 408 stands, it contains a direct contradiction of Hering's colour-theory (p. 407), which may lead the student astray. The paragraph on corresponding retinal points and on the horopter seem a little uncritical. The author is throughout specially happy in his diagrams. Fig. 223, p. 429, which illustrates the action of of the muscles of the eye-ball, deserves in particular to be copied into all future physiological and psychological text-books. E. Fick's replies (1889, 1890) to the investigations of van Genderen-Stort and Engelmann have apparently not been noticed (p. 425). For binocular contrast the reader is referred to Hering. This is an error: Fechner's elaborate discussion was published in 1860, and the first experiment quoted is taken from him.

Much that is psychophysical is included, again, under the rubric of "physical data" in the case of the ear. I am very glad to see that Dr. Waller roundly rejects the hypothesis that the semi-circular canals are concerned in the appreciation of the direction of sound. He proposes a modified form of Rutherford's theory of hearing (independently arrived at) in place of that of Helmholtz. What is wanted here is facts; and especially facts relating to the mode of vibration of the auditory hairs. Nothing is said of a theory of taste or smell, or of the temperature-sense: and the treatment of the "muscular sense" is unsatisfactory, no reference at all being made to Goldscheider's work on articular sensibility. The incorrect statement made in this connexion about Wundt has probably been taken from an early edition of the *Physiologische Psychologie*.

The two final chapters deal with the spinal cord and bulb, and with the brain. The evidence relating to sensory and motor conduction-paths in the cord is carefully sifted. Good remarks are made upon the "centres" of the cord and the medulla; as regards which the beginner (I can speak from experience)

is especially likely to go wrong. The possibility of the presence of retino-motor fibres in the N. opticus might have been alluded to. The discussion of the facts and hypotheses of cortical localisation is excellent. The student is impressed with the enormous complication of the subject, and with the need of extreme caution in drawing inferences, at the same time that data are furnished him for an appreciation of the value of conflicting methods and statements. The author recognises the unsuitability of Munk's nomenclature, and is praiseworthily careful in regard to Jackson's schema. In the paragraph on reaction-time, the influence of the direction of attention is noted; but it should then be stated that the cases given in fig. 279 illustrate the "muscular" form. The times adduced for touch and hearing are surely too long. In the statement of the Weber-Fechner law it should be insisted that the "least observable differences," or sensation units are only regarded by Fechner as equal in their subjective contexts, and in the absence of all opportunity for objective measurement or estimation. Dr. Waller plainly means this, but says it rather curtly; and there has been much misunderstanding of the point.

The explanation of the optical illusions on p. 541 is too short; it credits the reader with certain psychophysical knowledge. The question as to the nature of colour-contrast (for the answering of which Kirschmann's work should have been taken into account) is left just where Fechner left it thirty years ago. I do not mean this as a reproach; it may be that the answer given is correct; in any case it is, in this instance, based partially upon the author's own experiments. But one should certainly take some account of binocular contrast, in seeking a plausible hypothesis; and if one inclines, in whole or part, to a psychological interpretation of the phenomena, one should avoid the word "judgment." The chapter concludes with some very sensible paragraphs upon hypnosis.

So far as it has been here examined, this book is to be cordially recommended. The style is lucid and simple; and Dr. Waller handles "frontier questions" with a modesty and soundness which, if common, would soon put an end to the mutual distrust that, unfortunately, so often exists between the physiologist and the psychophysicist. It remains to be said that the rest of the work consists of seven chapters on the phenomena of nutrition; three appendices (on the origin and nutrition of the embryo, on constitutional formulæ of proximate principles, and on units of measurement); a confessedly incomplete, but useful bibliography; and a good index.

E. B. Titchener.

Contribution a l'étude anatomique et clinique de l'Acromégalie et en particulier d'une forme amyotrophique de cette maladie. Par le Dr. G. DUCHESNEAU, ancien interne des Hopitaux et de la Maternité de Lyon. Chap. vii., pp. 206. Paris: Baillière et Fils, 1892.

Dr. Duchesneau admirably justifies the title of his monograph: his contribution is truly a study, and a valuable addition to the now extensive literature of the subject. From the number of cases reported, and the careful analysis of symptoms, we can strongly recommend its perusal to all who wish to investigate the disease.

The first chapter summarizes the previous literature of Acromegaly, and briefly traces the gradual separation of this disease by Marie from Paget's osteitis deformans, Virchow's leontiasis ossea, myxoedema, and other diseases all having a strong family likeness. It is also mentioned that in 1890 Marie, from a careful examination of M. Gourand's case, recognized "hypertrophic pulmonary osteo-arthropathy" as a disease having a definite causation, and distinct from Acromegaly with which it had before been confounded. Dr. Duchesneau points out that although since Marie's original essay and M. de Souza Leite's thesis many clinical cases, reviews and anatomo-pathological papers have been published, most attention has been paid to the theoretical side of the question, and expresses his desire to treat rather of nosography and pathogeny.

The second chapter contains 31 collected cases, nearly all recorded since the publication of M. de Souza Leite's thesis; they are carefully and conveniently arranged into three groups, 1st (14 in number), comprising observations sufficiently complete for anatomical or clinical study; 2nd (13 in number), cases only shortly detailed; 3rd (4 in number), cases described as acromegaly, but not really examples of that disease. Of these latter the author considers that three were probably cases of hypertrophic pulmonary osteo-arthropathy, the fourth is rejected from the early age of the subject and the absence of any enlargement of the inferior maxilla.

The third chapter shortly refers to ætiology. With M. de Souza Leite the author admits how little is at present known of the causes of acromegaly but gives the following results of an analysis of the cases collected by himself, together with those of M. de Souza Leite.

That acromegaly is slightly more frequent among females than males, numbers being 30 and 23 respectively. That it is not congenital.

That nervous affections, including painful moral impressions and psychoses, may act as causes, "soit predisposantes, soit même déterminantes." Dr. Duchesneau remarks it is curious to note that in no case among his own collection, and only in three of M. de Souza Leite's, is there any history of syphilis or alcohol.

The fourth chapter treats of symptomatology. A rapid sketch is first given showing what is the type *le bloc* of acromegaly. Secondly, the groups of symptoms are given in detail and put in relief. Thirdly, Dr. Duchesneau's own case is given, the symptoms analysed and compared with those found in other cases. It is pointed out that there is at least a form of acromegaly which, from the striking alterations in muscles, may be qualified as "myopathique."

Dr. de Souza Leite's method is followed by Dr. Duchesneau in rapidly sketching the symptoms which are therefore classified, thus:—

1. Constant Objective Symptoms.—*Hypertrophy* of the *extremities* of upper and lower limbs, especially of the hands and fingers, feet and toes, all the tissues being implicated and giving an appearance which has been compared to paws or battledores.

The dimensions of the wrist and forearm are usually unaltered, those of the ankle and leg are less frequently normal. But as Marie has remarked the greatest change is seen in "*the bones of the extremities and the extremities of the bones.*" In the *head*, the cranium is little altered, thus forming a striking contrast to the face which is oval and elongated vertically. The nose, lips, especially the inferior and the lower jaw attract attention at once, all being immensely enlarged, the latter giving a massive prognathism to the face. The tongue is usually much enlarged.

In the *trunk* the spine and thorax are affected, the former presenting a kyphosis in the cervico-dorsal region from hypertrophy and deformity of the vertebræ; the latter projecting from deformity of clavicle sternum, ribs, &c., forms with the spine a "double hump" (Marie).

2. Inconstant Objective Symptoms.—An enlargement and thickening of the neck; an enlargement of the larynx; atrophy of the mammary glands; a pendulous condition of the abdomen; hypertrophy of the genitals; varicose veins; phosphaturia, albuminuria, &c., &c.

3. Constant Subjective Symptoms.—Marked cephalalgia; amenorrhea, without loss of sexual power. Alterations in special senses, especially sight. Exaggerated appetite and great thirst.

4. Inconstant Subjective Symptoms.—Alteration of hearing, smell, taste; palpitations of the heart; severe abdominal pains simulating colic.

5. General Symptoms.—Lassitude, disinclination to exertion. Mental depression. After giving at great length the symptoms of the head, the trunk, the limbs and the various systems, Dr. Duchesneau gives in detail his own case, and notes the points of agreement or disagreement with his fourteen collected cases as well as with the classical type of acromegaly. Dr. Duchesneau's case was under observation for fifteen years, and was an undoubted example of the disease, but is also exceedingly interesting and noteworthy, clinically and pathologically, for reasons of neurological and general interest. It is only possible here to enumerate the principal points: the onset, with acute pains in various regions, head, spine, limbs, &c., with loss of power; the presence of hypertrophy, obtaining in the usual situations; the continuance and gradual increase of the pains and amyatrophy, gradual loss of acute vision, passing attacks of œdema at the crisis of the pains; enlargement of the thyroid gland; the presence of a floating kidney; long-continued *phosphaturia;* final termination by nephritis. After carefully analysing all the symptoms Dr. Duchesneau draws special attention to the very advanced muscular atrophy, and quotes the cases of Godlee, Gabian and Claus to support his contention that there is an amyotrophic form of acromegaly. To the amyotrophy is attributed, partially at any rate, in addition to the loss of power in the limbs, the marked kyphosis, the chest deformity, and the floating kidney.

This long chapter, too long we think, is ended by a consideration of other clinical forms. Dr. Duchesneau points out the disproportion in various cases between the ocular troubles and the duration of the case. He does not agree with the hitherto generally accepted view that the hypertrophy of the hypophysis cerebri will always account for these troubles, and therefore suggests as another clinical form of acromegaly a type characterised by alterations of visual apparatus.

Finally, a juvenile form, with general increased growth, is mentioned, but not enlarged upon.

The fifth chapter treats of pathological anatomy, being an account of the lesions found in Dr. Duchesneau's case and their comparison with those which have obtained in other cases. The constant pathological lesions are termed the "*Triade hypermegalique*," viz.: lesions of the skeleton, integuments and hypophysis cerebri. There are also less constant lesions found in the

various systems. Dr. Duchesneau's case agreed in most points with the classical type, but there are several very important variations. Moreover, a microscopical examination of the bony tissues has elicited some striking facts bearing on the pathology.

The *cranium* presented more changes than usual. These Dr. Duchesneau attributes to the advanced stage of the disease. The sutures were almost obliterated; the parietal and occipital bones of uneven thickness; the superior maxilla much hypertrophied. The *spine* presented no lumbar lordosis. On opening the spinal canal there were no inflammatory adhesions of the dura mater, but the canal throughout was lined with a remarkably thick layer of "*cellulo-adipose*" tissue, which appeared to belong to the periosteum. It was in some places a centimetre in thickness; passing through the articular foramina of the vertebræ it compressed the nerves, which, even to the naked eye, were evidently altered. And it was just those nerves supplying the atrophied muscles, which were most altered. Microscopic examination of their roots showed disappearance of axis cylinders and segmentation of the myelin. These lesions thus account for the neuralgic phenomena and the muscular atrophy.

Dr. Duchesneau ably demonstrates by a consideration of sections taken from various bones that the increase in their size is always due to an increase in the spongy tissue, which has taken place at the expense of the compact tissue, so that there is a remarkable increase in the number and extent of the medullary spaces. This change takes place very gradually, and is not inflammatory, as after a prolonged and careful examination no giant cells, myeloplaxes, or Bizzozero's cells, with budding nuclei, were ever found. Therefore this condition differs from osteitis, and also from that of young growing bone; it differs also from rickets where the increase in size of the bone takes place in the cartilage, periosteum and outer compact tissue at the expense of the medullary portion. Again the changes in acromegaly are slow, regular and continuous, while in Paget's disease a tumultuous heaping up of the spongy tissue takes place, and isolated fragments of bony lemellæ are found, being the remains of broken-down Haversian systems. Lastly, Dr. Duchesneau remarks that all the bones affected normally contain red marrow, marrow, that is, remaining embryonic.

The changes described in the integuments are analagous to those mentioned above as found in the bony-tissues, viz., an increase of fibro-adipose tissue occurring slowly and without inflammation so that there is a real hyperplasia. The orifices of the sebaceous hair follicles were enlarged and their secretion had been increased. The nails were much striated. The increase in size of the tongue was due to increase of "*cellulo-adipose*" tissue; the muscles were not affected, there were no distended lymphatic spaces as in true macroglossia, the mucous membrane was healthy.

The hypophysis cerebri, owing to its fragile condition, could not be examined.

A systematic examination of the brain and cord has yet to be made.

There was a persistent thymus, normal in structure though hypertrophied. The enlargement of the thyroid was due to a cystic degeneration in some parts of which ossification had taken place; it closely resembled, under the microscope, an alveolar carcinoma. And attached to the first two ribs on the left side were two tumours of the same structure, each about the size of a fowl's egg.

Floating kidney was found as diagnosed and also nephritis accounting for death.

The sixth chapter contains some general observations on acromegaly. The principal theories which have been propounded are shortly referred to. Freund regards acromegaly as an *anomaly of development*. Klebs on the strength of a persistent thymus gland has constructed the theory of *l'angiomatose thymique*. Holschewnikoff and Recklinghausen elaborated a *nervous theory*, thinking that a lesion of the central and peripheral nervous system will account for the disease. Marie has connected acromegaly with *enlargement of the pituitary body*. Dr. Duchesneau does not accept any of these theories, there being a want of evidence in support of them. Returning to his own case he proceeds to connect the systematic hyperplasia of spongy tissue, in bones having red marrow, with the phosphaturia observed during life; and shows by a chart that at special crises, when fresh changes may be supposed to have been going on in the bones, there was an increase in the amount of phosphoric acid eliminated, thus indicating the onward match of the disease. Next Dr. Duchesneau suggests a relation between the bony lesions, the fibrous tissues inclosing them and the integuments surrounding both. He supposes that the connective tissue bundles of the periosteum when no longer taken up to form fibrous tissue as the fibres of Sharpey simply develop into ordinary fibrous tissue, that the blood vessels of the periosteum, no longer used to supply compact growing bone degenerate into fatty columns. Thirdly several factors are suggested as taking part in the hyperplasia of the integuments. A stretching of the tissues by the enlargement of the bones. A hyperplasia from chronic venous and lymphatic congestion, &c. In chapter seven various methods of past treatment are reviewed and the opinion expressed that in the present state of our knowledge treatment can only be symptomatic.

From this brief resumé it will be seen at once that the value of Dr. Duchesneau's exact and careful observations needs no accentuation. The changes noted in bony tissue doubtless occur in all cases of acromegaly, and the amyotrophic form is well accounted for by the nerve changes found in Dr. Duchesneau's own case. The cellulo-adipose tissue derived from the periosteum in its turn seems to have caused the nerve lesions. Thus the chain of events is complete so far, and we hope, with the author,

that other parts of the pathogeny of acromegaly will in like manner soon be cleared up.

We await with interest the report of the systematic examination of the brain and spinal cord, which has yet to be made. The author makes no mention of the sympathetic nervous system, the hypertrophy of which was regarded by Marie as being absolutely constant in acromegaly, but perhaps he will report on this also with the other systems. It is important that this should not be overlooked in future autopsies.

The possible part played by the amyotrophy as a factor in the production of the bony deformities, kyphosis, talipes, &c., and also by relaxing abdominal pressure of the dislocation of the kidney is an interesting theory, but we think Dr. Duchesneau has not quite established it. For kyphosis, with deformity of the vertebræ, is a constant symptom, while amyotrophy, at any rate a considerable amount of it is inconstant. Again, floating kidney is fairly common in women, especially in multipara, although laxity of the abdominal walls is doubtless a factor in them, there is no large amount of muscular atrophy.

Dr. Duchesneau advances good grounds for recognizing the existence of a form of acromegaly with prominent ocular troubles, and expresses his opinion that the mechanical action of the enlarged hypophysis is not the sole factor in its production, although he does not suggest any others. Possibly the narrowing of the optic foramina, caused like that of the vertebral foramina by bone changes and compression by the periosteal cellulo-adipose tissue, may be one of the factors in such cases.

With regard to the general aetiology we would advise caution in accepting, without weighty evidence, depressing nervous influences, the psychoses, &c., as causes, since histories of such troubles are not only very common in various diseases but so easily obtained that not much reliance can generally be placed on them. The presence of mental depression in acromegaly is often explained we think by the pressure of the tumour at the base of the brain.

Lastly, treatment so far can be only symptomatic, but may not the phosphaturia which probably varies directly with the rate of degeneration in the bones at some future time be a guide to treatment. If not itself to be treated it will possibly indicate the rapidity of the disease and thus aid in its regulation.

Meanwhile Dr. Duchesneau is to be congratulated on his very excellent monograph and the progress made in the pathology of acromegale from his careful investigations. As Virchow has said "we are here engaged on a most obscure subject which probably only in the course of several years will be perfectly elucidated through new observations." And it is by such accurate and extended observations as Duchesneau has made that we may expect to finally clear up the subject.

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